



1993

Evaluation and Treatment of Shoulder Impingement Syndrome in the Absence of Rotator Cuff Tear

Pat R. Carter
University of North Dakota

Follow this and additional works at: <https://commons.und.edu/pt-grad>



Part of the [Physical Therapy Commons](#)

Recommended Citation

Carter, Pat R., "Evaluation and Treatment of Shoulder Impingement Syndrome in the Absence of Rotator Cuff Tear" (1993). *Physical Therapy Scholarly Projects*. 90.
<https://commons.und.edu/pt-grad/90>

This Scholarly Project is brought to you for free and open access by the Department of Physical Therapy at UND Scholarly Commons. It has been accepted for inclusion in Physical Therapy Scholarly Projects by an authorized administrator of UND Scholarly Commons. For more information, please contact zeinebyousif@library.und.edu.

EVALUATION AND TREATMENT OF SHOULDER IMPINGEMENT
SYNDROME IN THE ABSENCE OF ROTATOR CUFF TEAR

By

Pat R. Carter
Bachelor of Science in Physical Therapy
University of North Dakota, 1980



An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of the requirements

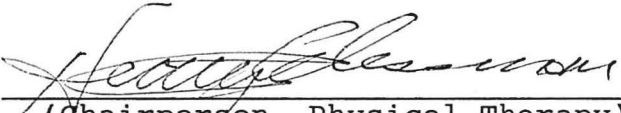
for the degree of

Master of Physical Therapy

Grand Forks, North Dakota

May
1993

This Independent Study Report, submitted by Pat R. Carter in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Chairperson of Physical Therapy under whom the work has been done and is hereby approved.


(Chairperson, Physical Therapy)

PERMISSION

Title Evaluation and Treatment of Shoulder Impingement
 Syndrome in the Absence of Rotator Cuff Tear

Department Physical Therapy

Degree Master of Physical Therapy

In presenting this independent study report in partial fulfillment of the requirements for a graduate degree from the University of North Dakota, I agree that the library of this University shall make it freely available for inspection. I further agree that permission for extensive copying for scholarly purposes may be granted by the professor who supervised my independent study work or, in his absence, by the Chairperson of the department or the Dean of the Graduate School. It is understood that any copying or publication or other use of this independent study report or part thereof for financial gain shall not be allowed without my written permission. It is also understood that due recognition shall be given to me and to the University of North Dakota in any scholarly use which may be made of any material in my independent study report.

Signature Pat R. Carter

Date March 10, 1993

TABLE OF CONTENTS

LIST OF ILLUSTRATIONS.....	v
LIST OF TABLES.....	vi
ACKNOWLEDGEMENTS.....	vii
ABSTRACT.....	viii
INTRODUCTION.....	1
Anatomy.....	4
Biomechanics.....	10
LITERATURE SEARCH - ETIOLOGY	
Anatomical and Mechanical Causes.....	15
Vascular Causes.....	20
Traumatic Causes.....	23
Degenerative Causes.....	28
CLINICAL EVALUATION	
Subjective Evaluation.....	31
Objective Evaluation.....	33
TREATMENT.....	46
Initial Phase.....	47
Rehabilitation Phase.....	52
Prevention.....	67
Surgical Management.....	70
CONCLUSION.....	72
REFERENCES.....	76

LIST OF ILLUSTRATIONS

Figure	Page
1. Schematic representation of 4 joints comprising the shoulder.....	5
2. Anatomy relative to impingement syndrome.....	7
3. The coracoacromial ligament viewed laterally.....	8
4. Rotator cuff tendon insertion to humerus.....	11
5. Tests for impingement.....	41
6. Anterior instability test followed by relocation test.....	43
7. Posterior capsule stretching.....	54
8. Scapular plane elevation (scaption) in internal rotation.....	58

LIST OF TABLES

Table	Page
1. Initial Treatment Phase - Goals and Methods.....	48
2. Rehabilitation Treatment Phase - Goals and Methods.....	48

ACKNOWLEDGEMENTS

The editor expresses sincere gratitude and appreciation to Henry C. Wessman J.D., P.T., Chairman of the Department of Physical Therapy, for his assistance and advice in the preparation of this independent study.

ABSTRACT

Shoulder impingement syndrome is believed to be the most common cause of shoulder pain in adults, and the major shoulder problem in sports medicine. It develops in a progressive manner to bind the rotator cuff and subacromial bursa beneath the coracoacromial arch superiorly and the upper humerus inferiorly. If left untreated, rotator cuff degeneration and eventual partial or complete tears of the tendons may result.

This report focuses on stage 1 and 2 of impingement syndrome, excluding calcific tendinitis and known rotator cuff or biceps tears. Through a search of the literature, normal and pathologic anatomy and biomechanics will be examined. Rotator cuff vascularity, abnormal shoulder anatomy and biomechanics, trauma, and degeneration are etiologic factors which are discussed. These may be solely or jointly responsible for the symptoms and dysfunction produced by impingement syndrome.

A thorough evaluation is necessary to diagnose shoulder impingement syndrome. General shoulder evaluation procedures are performed, along with special tests for impingement and instability. Once diagnosed, early intervention is crucial

in the treatment of this problem. Conservative management is highly successful in stage 1 and 2 and may consist of medications, rest, physical modalities, sport or activity modification, exercise and prevention. The physical therapist is skilled in evaluation of this condition, and can work with the physician and patient to develop a rehabilitation program.

The exercise prescription initially may include isometrics, but will advance to isotonics, use of resistive equipment, and possibly isokinetics. Special attention is paid to strengthening and balancing the shoulder internal and external rotators. Considering the rotator cuff vascularity, EMG studies, and patient comfort, rotational exercises may be more effective if done with the arm positioned in slight flexion and abduction. The scapular rotators are also vitally important in rehabilitation of shoulder impingement syndrome. A majority of patients will respond to conservative, nonoperative efforts and will be able to resume sport or job activities.

EVALUATION AND TREATMENT OF SHOULDER IMPINGEMENT SYNDROME IN THE ABSENCE OF ROTATOR CUFF TEAR

Impingement syndrome (IS) has been cited as being the most common cause of shoulder pain in adults,¹ and the most common shoulder problem in sports medicine.² The diagnostic phrase, impingement syndrome, was popularized by Neer^{3,4} in 1972 when he recognized impingement of the rotator cuff as one of the causes of chronic shoulder disability. Impingement syndrome has also been referred to as degenerative tendinitis, subacromial or subdeltoid bursitis, and supraspinatus tendinitis⁵. Depalma⁶ uses the term painful arc syndrome synonymously with IS. These diagnostic phrases allude to a condition where there is binding of the rotator cuff and subacromial bursa beneath the coracoacromial arch above and the upper humerus below.¹ A more inclusive definition of impingement is provided by Matsen and Arntz^{7(p623)} as "the encroachment of the acromion, coracoacromial ligament, coracoid process, and/or acromioclavicular joint on the rotator cuff mechanism that passes beneath them as the glenohumeral joint is moved, particularly in flexion and rotation." The tendon of the long head of the biceps may also be stressed by these same structures and is often included in painful arc syndrome or IS.⁸

Individuals who use their arms in a position at or above horizontal are susceptible to impingement. Such occupations may include mill work, carpentry, tree pruning, fruit picking, nursing, grocery clerking, longshoring, warehousing, and painting.^{1,7,8} Competitive and recreational athletes who repeatedly use their arms above the horizontal may also develop IS. Participants involved in swimming, tennis, and the throwing sports of baseball pitching or quarterbacking are prone to this.^{1,9} However, IS is not isolated to certain job activities or sports, as sedentary individuals may also develop IS. One-third of the new patients with cuff tendon involvement at the University of Washington Shoulder Clinic had no specific precipitating factor to account for their IS symptoms.⁷ Any age group may present with impingement; from teenagers to middle-age workers, and weekend athletes to the elderly patient. The most frequently noted onset is in individuals aged 50-59 years.^{1,6} Depalma⁶ gives further epidemiologic information gained from his clinical experience. He believes that men are more afflicted than women with a ratio of 3:2 and the right shoulder is involved twice as often as the left.

The IS patient may present with subjective symptoms of gnawing, aching pain over the deltoid insertion area. Activities involving overhead positions of the arms will aggravate symptoms, and activities of daily living such as pouring coffee or curling and combing hair will also produce

pain. Night pain may be a pronounced feature, with the patients reporting difficulty sleeping on the involved shoulder.¹⁰ Common objective findings include a painful arc of motion between 60 and 120 degrees, limited range of motion, pain with forward flexion, tenderness over the cuff and acromion, possible atrophy of the deltoid and cuff muscles, and pain with resisted strength testing of the shoulder muscle groups.^{6,7}

In 1983, Neer¹¹ described three stages of IS which are progressive in nature. Stage 1 is edema and hemorrhage. This usually occurs in patients aged 25 or younger, but can be present in the 20-40 age group of weekend athletes who have not had chronic symptoms.¹ A key point of stage 1 is that it is generally a reversible lesion.⁹ Stage 2 is fibrosis and tendinitis. The bursa becomes fibrotic and thickened, which magnifies the problem.¹¹ This commonly occurs in the athlete or worker in the 25-40 age range. Fibrosis and degeneration of the supraspinatus and biceps tendon may also occur. The key points of stage 2 are pain with activity, a prolonged history, and increasing difficulty in attempts to reverse the lesion.^{9,12} Stage 3 demonstrates tears of the rotator cuff, biceps ruptures, and bone changes. Stage 3 has been defined by some authors to include tears of less than 1 cm, with stage 4 the tears of 1 cm or larger.^{2,13} Stage 3 and 4 of IS occurs almost entirely after

the age of 40 and may cause progressive disability. The key point of this stage is tendon degeneration.⁸

Although IS can be a progressive disorder of the shoulder, there is much agreement that it responds well to conservative treatment and nonoperative management.⁵⁻⁸ It has been stated that 80% of patients will benefit from the use of conservative measures.¹ The role of physical therapy in the management of IS is an important aspect in the overall evaluation and treatment of these patients; whether young athletes, middle aged workers, or retirees.

The focus of this paper will be on stage 1 and 2 of IS, excluding calcific tendinitis and known rotator cuff or biceps tears. Normal and pathologic anatomy, etiology, clinical evaluation and conservative management approaches are topics to be covered within this context. A special emphasis will be placed on the role of physical therapy in the rehabilitation of patients with IS.

ANATOMY

The shoulder joint is one of the most complex articulations in the body with 3 bones, 4 joints, 12 ligaments and over 15 muscles. This complex arrangement allows the glenohumeral joint to be the most mobile in the body with the humerus capable of being moved through a space exceeding a hemisphere.¹⁰ Figure 1 is a schematic representation of the 4 joints comprising the shoulder complex. With increased

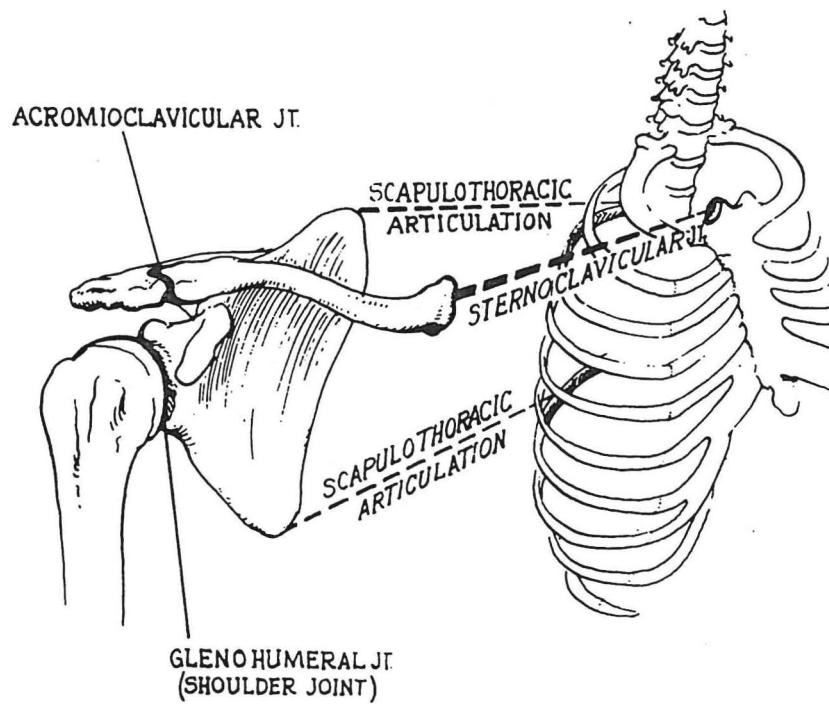


Fig 1.--Schematic representation of 4 joints comprising the shoulder. (From Hoppenfeld S. Physical Examination of the Spine and Extremities. New York, NY: Appleton-Centruy-Crofts; 1976:p 2.)

mobility, an accompanying loss of stability may pose problems, especially for the athlete, where excess mobility has resulted from external rotation, anterior humeral head displacement, high velocity, force and repetition.¹⁴ The nonathlete population relies on the shoulder complex along with other upper extremity joints and muscle groups to place and control the position of the hand in the visual work space in front of the body.¹⁵ The function of the shoulder, whether simple or complex, is compromised when the symptoms of IS are present.

The anatomy relative to IS is shown in Figure 2 and 3. It includes the upper surface of the humerus, the rotator cuff tendons, the long head of the biceps, the acromioclavicular (AC) joint, the subdeltoid or subacromial bursa, and the coracoacromial arch. The coracoacromial arch consists of two scapular processes - the coracoid and the acromion which are connected by the strong, triangular shaped coracoacromial ligament (CAL).⁷ Although not a true articulation, the subacromial region has been described as the "second joint" of the shoulder - a functional, extracapsular joint between the coracoacromial arch and the greater tuberosity.¹⁶

Progressing from superior to inferior in this anatomic area, the AC joint may be described first. This is a freely moveable synovial joint between the lateral end of the clavicle and the anterior medial aspect of the acromion process of the scapula.¹⁷ According to Ogata and Uhthoff,¹⁸

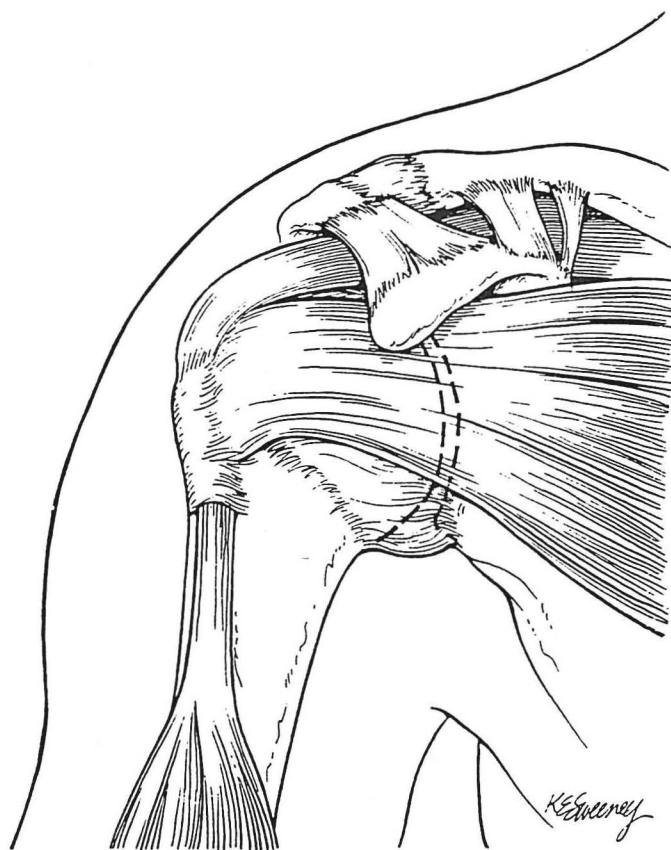


Fig 2.--Anatomy relative to impingement syndrome. (From Matsen FA, Arntz CT. Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The Shoulder. Philadelphia, PA: WB Saunders Co; 1990:p 624.)

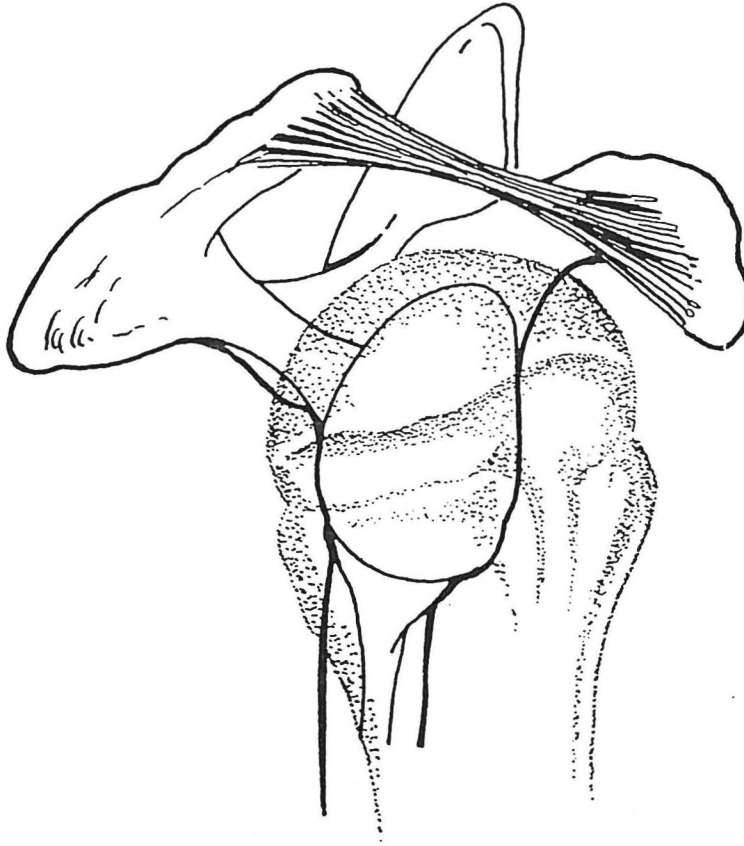


Fig 3.--The coracoacromial ligament viewed laterally. (From Peat M. Functional anatomy of the shoulder complex. Phys Ther. 1986;66:p 1860.)

the cortex of the undersurface of the acromion should line up with the medial distal end of the clavicle. This was the case in 43% of 76 cadaver specimens and was considered a normal finding. The CAL, which passes upward, laterally and slightly posteriorly from its base at the lateral coracoid process, was found to attach to the anteromedial border of the acromion with its superficial fibers only. The majority of fibers attached to the undersurface of the acromion in a crescent shape. Fibrofatty tissue covers the remaining undersurface. This normal configuration provides a smooth concavity of the subacromial gliding mechanism. The subacromial bursa blends with the CAL and acromion and extends beneath the deltoid muscle and the coracoid process of the scapula.¹⁹ The bursa adheres to the rotator cuff from below. It does not normally communicate with the joint and is important for allowing gliding between the acromion and the deltoid and rotator cuff muscles.^{15,20} The bursa is not actually a space, but rather two serosal surfaces lubricated by synovial fluid.^{7,19}

Beneath the subacromial bursa lies the rotator cuff. It is the musculotendinous complex formed by the attachment to the capsule of the supraspinatus superiorly, the infraspinatus and teres minor posteriorly and the subscapularis anteriorly.¹⁵ The supraspinatus tendon inserts at the superior aspect of the greater tuberosity after it has crossed over the top of the humerus at an angle of 90° from its origin.²¹

Figure 4 demonstrates the tendon insertions and the close proximity of the supraspinatus tendon and the coracoacromial arch. The infraspinatus tendon inserts at the middle aspect of the greater tuberosity, the teres minor at the inferior aspect of the greater tuberosity, and the subscapularis tendon at the lesser tuberosity.¹⁷ These four tendons are blended and fused together as they cross the joint prior to insertion on the humerus. The cuff itself is continuous in the form of a sheath around the biceps tendon.^{7,15} The long head of the biceps tendon originates at the supraglenoid tubercle and penetrates the rotator cuff between the subscapularis and supraspinatus tendons. The tendon lies in the bicipital groove; a vertical groove between the greater and lesser tuberosities which is superficially closed by the transverse ligament.¹⁹

BIOMECHANICS

Neer⁴ emphasized that the arm is most often used in an anterior and often internally rotated position. Other authors have supported the belief that the functional arc of motion for the shoulder occurs in the anterior plane.^{8,9,21} When the arm is elevated forward, the supraspinatus passes under the anterior edge of the acromion and the AC joint.¹¹ Forward flexion with internal rotation causes the supraspinatus tendon and greater tuberosity to articulate close to the undersurface of the anterior acromion.¹² When abduction

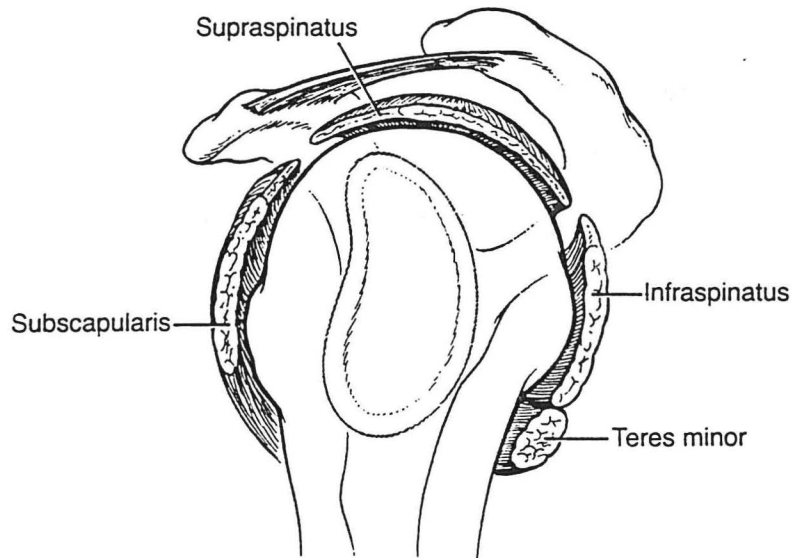


Fig 4.--Rotator cuff tendon insertion to humerus. (From Matsen FA, Arntz CT. Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The Shoulder. Philadelphia, Pa: WB Saunders Co; 1990:p 624.)

is performed, external rotation must occur to depress the greater tuberosity beneath the acromion in order that the tuberosity does not abut against the acromial arch.²¹ When the arm forward flexes or abducts, it is the subacromial bursa which allows smooth gliding motion between the rotator cuff structures and the coracoacromial arch.¹²

To enhance smooth, coordinated movement of the glenohumeral joint into flexion or abduction, the prime movers acting on the humerus must function with other stabilizer muscles to avoid producing a subluxating force and resultant instability.¹⁵ It is the force couple between the deltoid and rotator cuff muscles which applies the force necessary to elevate or abduct the arm. The force couple is produced by the effects of opposing forces between the deltoid acting below the center of rotation, and the short rotators acting above the center of rotation.¹⁵ Bechtol²² explains that the deltoid, which tends to displace the humeral head from the glenoid, is counteracted by the rotator cuff which fixes the humeral head in the glenoid.

The size and leverage of the deltoid gives it the ability to elevate the arm independently. However, at the beginning arc of movement, the deltoid functions less efficiently due to the low angle of pull. This reduced leverage makes upward displacement of the humeral head the dominant effect.¹⁹ The deltoid becomes more efficient as elevation continues. The required lifting force at 30° of abduction is 50% of

maximum and decreases to 43% at 90° and to 18% at 150° .

If the deltoid is absent, but the rotator cuff muscles intact, about 50% of abduction power is present through the range of motion.²²

The supraspinatus has been identified as an initiator of abduction, but acting alone, it would have to contract at 97% maximum strength to lift the arm the first 30° .¹⁹ Almost all of the supraspinatus' force is directed toward compression which makes its major function one of joint stabilization. When the supraspinatus is absent but the joint free of pain, abduction can be initiated with approximately 80% of normal power. This power rapidly falls off however, and by 90° the weight of the arm can barely be lifted against gravity, although full range of abduction can be accomplished.²²

The angle of pull of the infraspinatus and subscapularis is estimated to be downward at 45° and the teres minor slightly more vertical at 55° .¹⁹ During arm elevation, the remaining three rotator cuff muscles exert an equal force into downward displacement of the humeral head. The biceps also acts as a depressor of the humeral head and restraining force against upward migration.¹¹

To summarize the force couple coordination: (1) the deltoid supplies the dominant force to rotate the arm upward with the supraspinatus reducing the deltoid demand, and (2) the shearing force of the deltoid to displace the humerus

vertically is opposed by the weight of the arm and active depression of the infraspinatus, subscapularis and teres minor. The rotator cuff muscles function together to provide joint compression throughout the range of elevation. Once the arm is above horizontal, the rotator cuff action decreases while the deltoid continues its activity. Pink and Jobe¹⁴ also emphasize the importance of the scapular rotators (trapezius, levator scapulae, rhomboid, serratus anterior, and pectoralis minor) and the other muscles acting on the humerus (clavicular pectoralis major and latissimus dorsi) for efficient, coordinated arm elevation. In addition to this force couple, normal shoulder function is also dependent on normal capsular laxity and adequate subacromial space.⁷

LITERATURE SEARCH - ETIOLOGY

Anatomical and Mechanical Causes

Because limited space is available for the rotator cuff tendons to pass under the coracoacromial arch, impingement may occur if: 1) there is an increase in volume of the structures passing beneath the coracoacromial arch, or 2) there is a decrease in the space allowed for the rotator cuff and biceps tendons.^{9,23,24} Neer¹¹ believes that mechanical impingement is the cause of 95% of rotator cuff tears. He described the mechanical impingement of 11 dissected scapulae as "having a characteristic ridge of proliferative spurs and excrescences on the undersurface of the anterior process ... apparently caused by repeated impingement of the rotator cuff and humeral head. ..." ⁴(p41) The spurs and outgrowths on the undersurface of the acromion reported by Neer⁴ decreases the space available for the cuff tendons.

Other authors have examined the inferior aspect of the acromion and its relation to IS. In a study of 200 cadaveric shoulders by Ozaki et al,²⁵ attritional lesions of the CAL and anterior one-third of the acromion undersurface were observed in specimens with incomplete bursal side tears and those with full thickness tears. Of the 23 specimens with incomplete bursal side tears, the undersurface of the acro-

mion was intact. On radiograph, 82 of 104 normal specimens had a regular trabecular pattern without sclerosis, hypertrophy or cystic changes and the remaining 22 had either spurs or osteophytes. In the specimens that had a torn cuff, every one had trabecular irregularity, sclerosis, hypertrophy, or cystic changes, mainly at the anterior aspect of the acromion. Based on the findings of their histological and radiographic results, Ozaki and associates²⁵ supported the concept that the anterior one-third of the acromion works as a subacromial joint and concluded that the pathogenesis of rotator cuff tears is due to a degenerative process.

In contrast, Ogata and Uhthoff¹⁸ found nearly one-half of the 21 specimens without incomplete or full thickness tears had a degenerated undersurface of the acromion. They did report that 86% of the incomplete articular side tears and all those with full thickness tears had an abnormal undersurface. A downward projection of the anteromedial part of the acromion in an area limited to the insertion of the CAL, was classified as a beak-type acromion. Based on roentgenograms, 57% of specimens with the beak showed evidence of degeneration of the acromion's undersurface, with only 27% of specimens with normal roentgenograms having acromial beaks. They suggested that the beak of the acromion could be a primary bony cause of narrowing of the subacromial compartment, resulting in impingement.¹⁸ A hooked acromion shape on roentgenogram was related to 41% of the 200 patients

studied by Bigliani.²⁶ Eighty percent of the patients with full-thickness rotator cuff tears confirmed by arthrogram, occurred in patients with hooked (Type III) acromion. Of the patients with stage 1 and 2 IS as diagnosed by physical examination, positive impingement test and negative arthrogram, there was no association between the type of acromion and the diagnosis.

The question these research studies bring up is whether the morphology of the acromion is a cause of impingement, a result of impingement, or both. Other structural factors of IS involving the acromion include unfused acromion, non-union or malunion.⁷

The role of the CAL in IS has also been found to be a factor, especially in the chronic stages.²⁷ When the CAL was studied with electron microscopy following surgery in 11 patients with chronic IS, the architecture of the ligament was found to be distorted when compared to 2 normal CALs. There was marked irregularity in size and shape of the fascicles, excessive infiltration by fibrofatty tissue into the ligament, and disarray in the arrangement of cells and the matrix. Sakar et al²⁷ found the cells to be metabolically active due to the strain induced on the ligament, but concluded that the CAL was not primarily responsible for initiating the process of IS.

Petersson and Gentz²⁸ reported instances during surgery for acromioplasty that no bony excrescences were found on

the undersurface of the anterior acromion. They did find distally pointing AC osteophytes which they believed to compress the supraspinatus tendon against the humeral head in abduction, causing chronic impingement of the tendon. Of 140 cadaver shoulder specimens investigated, 24% (40 shoulders) were found to have distally pointing osteophytes at the AC joint. They appeared in 29 shoulders with degenerative lesions or ruptures of the supraspinatus tendon and in 11 shoulders with normal tendons.

The coracoid process may also serve to decrease the space available for the rotator cuff at the anterior border of the subacromial space. In 1985, Gerber, Terrier, and Ganz²⁹ did CT scans of 56 shoulders and measured the distance between the coracoid tip and humeral head. The distance averaged 8.6 mm with the arm at the side, and decreased to an average of 6.7 mm with forward flexion combined with medial rotation. The authors noted that patients with proven subcoracoid impingement had symptoms of dull pain in front of the shoulder, at times extending to the upper arm and forearm, with pain reproduced by forward flexion and medial rotation. Coracohumeral impingement could be related to traumatic causes and idiopathic causes such as a larger than normal coracoid process which is more laterally placed.²⁹ Iatrogenic pathology resulting after shoulder instability surgery was also responsible for some cases of this type of impingement. In fact, Patte²³ believed up to 20% of poor results after

anterior acromioplasty were caused from this type of impingement. An increase in the contents of the coracohumeral space was found to occur more frequently than a decrease in size of the space. He related this to isolated traumatic tears of the subscapularis, usually accompanied by dislocation of the long head of the biceps.

Cephalic displacement of the humeral head is also another cause of a decrease in the subacromial interval.^{6,7} In the presence of a weakened cuff, the humeral head is unable to be stabilized in the glenoid and contraction of the deltoid will cause proximal migration of the humeral head. This disruption of the normal force couple between the rotator cuff and deltoid with upward displacement of the humeral head may then squeeze the remaining cuff against the coracoacromial arch. The balance of the individual rotator cuff muscles may also be upset in IS. When the ratio of internal to external rotators was measured isokinetically in athletes with IS, they were found to have significantly higher ratios than either athletes with instability or normal subjects.³⁰ The authors felt this represented a relative weakness of the external rotators in this group of athletes.

Hypertrophy of the soft tissues in the subacromial space may increase the volume and contribute to IS. Strains of the CAL may have resulted from volume changes in the soft tissue including the subacromial bursa, supraspinatus tendon and biceps tendon.²⁷ Repeated microtrauma may cause an in-

flammatory response with edema and an increase in volume of the tendinous structures.^{9,24} The increase in volume of the tendons and bursa is also interrelated to the vascular, traumatic and degenerative causes of IS.

Vascular Causes

Vascularity of the rotator cuff is a factor in IS. As early as 1934, Codman³¹ referred to a critical zone in the tendon of the supraspinatus where rupture commonly occurred. This area, located approximately 1 cm from the insertion, has been examined by researchers studying the vascular pattern of the rotator cuff. In 1963, Moseley and Goldie³² found 32 cadaver shoulder specimens had a rich vascular supply to the rotator cuff with no difference between age groups. They concluded the critical zone corresponded to an anastomoses between osseous and tendinous vessels. Bateman^{5(p249)} agreed with Moseley's findings stating "the area is anything but an avascular or ischemic zone" due to the insistent oozing when pricked with a needle or when resected during surgery.

In contrast to these findings, Rothman and Parke³³ did histologic studies and reported a paucity of arteries in the area of the supraspinatus just medial to the insertion in 63% of the shoulder specimens examined. They found the cuff tendons to be supplied chiefly by the suprascapular, anterior and posterior circumflex humeral arteries and second-

arily by the thoracoacromial, suprahumeral and subscapular arteries. Rathbun and Macnab³⁴ used the technique of microangiography carried out on cadavers as soon after death as possible, to determine the significance of the vascularity in the critical zone. They discovered the supraspinatus to have a constant area of avascularity related to its point of insertion. The infraspinatus occasionally had an area of avascularity, but the other tendons had good filling of the vascular bed. When the investigators abducted the opposite arm which had also been injected with micropaque solution, nearly complete filling of the vessels occurred throughout the tendon up to the point of insertion. They proposed it was possible that the constant pressure exerted by the head of the humerus on the supraspinatus tendon with the arm at the side could "wring out" the vessels in this area. The infraspinatus insertion and intracapsular portion of the biceps tendon were included with the supraspinatus when they theorized that the avascular zone preceded the degenerative changes.

In another study in 1989, Lohr and Uhthoff³⁵ found an area of hypovascularity in the supraspinatus tendon of 18 anatomic specimens evaluated, extending from 5 mm proximal to the bony insertion to the nearest musculotendinous junction. The size of the hypovascular area was not dependent on the age of the specimen. They confirmed Rothman and Parke's conclusion that the suprascapular artery was the

primary supplier of the supraspinatus tendon and that the rotator cuff was well supplied by the anterior and posterior circumflex anastomosis along with the thoracoacromial, supra-humeral and subscapular arteries. The articular side of the tendon was seen to be at a disadvantage in terms of blood supply than the bursal side which coincided with clinical findings of larger numbers of articular side incomplete lesions.

The vascular relationship to IS and rotator cuff degeneration has been challenged by some investigators. Matsen and Arntz⁷ speculate that the "wringing out" theory is not able to produce ischemia of sufficient duration necessary to cause tendon damage due to the frequent movements of the shoulder away from the side of the body. Lohr and Uhthoff's³⁵ speculation that the articular side of the tendon was more susceptible to degeneration and possible failure due to its diminished vascularization was questioned by Chansky and Ianotti.³⁶ They argue whether the degenerative changes seen in cadaver specimens is part of the normal aging process or a distinct clinical pathologic process. They also note a lack of correlation to clinical symptoms.

In addition, clinical observations made during surgery of hyperemic rotator cuff tendon tissue in the impingement zone has not corresponded with the cadaver arteriographic studies. A recent in vivo study using laser Doppler flowmetry was carried out by Swiontkowski et al.³⁷ Intraoperative

blood flow measurements were taken at multiple points along the subacromial surface of the rotator cuff in 15 patients aged 39-68 years. Of the four patients with tendinitis without partial or complete tear, all had significant blood flow within the tendon. Of the 11 patients with partial or complete tears, 9 had a hyperemic response. From the preliminary data and knowledge that there is a lack of baseline data on normal rotator cuffs, the authors concluded that: impingement may produce a hyperemic response within the rotator cuff tendon, the "avascular zone" was not identified and does not play a role in the pathophysiology, and the hypovascularity noted in the cadaver studies was probably an artifact of injection technique. Chansky and Iannotti³⁶ state that this implies hypervascularity or neovascularization is associated with symptomatic rotator cuff disease secondary to mechanical impingement. They believe the critical zone hypovascularity seen in the cadaver studies has a role in age related asymptomatic cuff degeneration. Whether or not these two processes are the same is unresolved.

Traumatic Causes

The traumatic causes of IS can be either classified as macro or microtrauma. Macrotrauma, including displaced fracture of the greater tuberosity, is representative of a rotator cuff tear (stages 3 or 4).³ Macrotrauma to the rotator cuff may also occur in an older person following

anterior dislocation. One report discussed 31 patients over 35 years old who were unable to abduct the involved arm after primary repair of anterior dislocation without fracture of the tuberosities. These patients were found to have a ruptured rotator cuff upon arthrography with the diagnosis being made an average of 7.6 months after surgery.³⁸

Microtrauma in the athletic population results in overuse syndromes related to repeated use of the upper extremity during sports.³⁹ Sports in which athletes are susceptible to IS include swimming, tennis, baseball pitching and quarterbacking, javelin throwing and gymnastics.^{14,39} Explosive, dynamic, and static forces provide the mechanism of microtrauma and may lead to IS.²⁴

Explosive forces are seen in the throwing sports when a maximal force is required for a brief period of time. Microtrauma ususally occurs at the musculotendinous junction or critical zone of the supraspinatus.²⁴ Of the four stages of throwing (windup, cocking, acceleration and deceleration) the deceleration stage has been indicted as being responsible for most of the rotator cuff stress.⁴⁰ During this stage, eccentric contraction of the posterior cuff produces a traction injury which may occur in the hypovascular zone. Accumulative stress also plays an important part of overuse injuries in baseball. There may be as many as 100 to 150 pitches in a game, all requiring explosive forces.

Tennis also requires explosive and repetitious forces. The racquet serves as an extension of the forearm and has the effect of increasing angular momentum by both increasing the force imparted to the ball and increasing forces needed to decelerate the arm during follow-through.³⁹ The serve and forearm stroke both have a cocking, acceleration and deceleration phase similar in ways to the baseball pitch. By cocking the arm to 90° , the deltoid provides compressive forces to the joint for stabilization. Further protection to joint structures is supplied by the external rotators (infraspinatus and teres minor) to maintain humeral head alignment and the subscapularis to limit excessive external rotation. The chance for acromial arch impingement is increased with overhead reaching when the arm is extended unless there is good external rotation.¹⁹

Swimming is also a sport of repetitious activity requiring dynamic forces at the shoulder. Shoulder impingement is the leading cause of shoulder problems in swimmers, with the free-style and butterfly swimmers most susceptible.^{21,41} Shoulder motion in swimming differs from that in baseball and tennis because the degree of external rotation motion and the velocity of internal rotation are less. However, a competitive swimmer will perform many more repetitions of shoulder motion each year than either the baseball or tennis player.³⁹

Static forces are required in sports such as gymnastics and weight lifting. Isometric contractions are needed for controlled movements such as involved in a ring routine or when weights are maintained overhead. In addition, gymnastics requires the shoulder provide momentum to sustain rapid compressive and distractive forces and also power to accelerate the body into various positions.^{24,39}

Athletes are not the only individuals prone to develop IS as an overuse disorder. At one shoulder clinic, one-third of the patients with cuff tendon involvement related the onset of their symptoms to their work.⁷ The repetitive, overhead tasks appear to be most responsible for causing IS in occupations such as carpentry, painting, and grocery clerking.

Penny and Welsh²⁴ state that whatever the mechanism, microtrauma establishes an inflammatory response which increases the volume of the soft tissue by edema formation, and increases the susceptibility of the area to further damage. The pain from IS can perpetuate a vicious circle by inhibiting muscle action resulting in rotator cuff weakness from disuse. Then when the normal force couple is lost, the rotator cuff is not able to counterbalance the vertical force of the deltoid which can further compress the soft tissues by proximal migration of the humeral head.¹⁹ Other authors also believe the mechanism of injury is complimentary to vascular causes resulting from mechanical impingement

in the vulnerable avascular region which is stressed by activity or task repetition.^{24,42}

Microtrauma and its resultant pathology has been linked to more than just IS. Jobe^{14,43} and other authors^{30,40} have noted the relationship between IS and shoulder instability in athletes. In this theory, subtle instability and traction to the rotator cuff sets up a continuum of the same disease which is progressed from instability to subluxation to impingement to rotator cuff tear.¹⁴ Impingement in this case has been termed secondary impingement and is defined as a relative decrease in the supraspinatus outlet related to instability of the glenohumeral joint.⁴⁴ This is most commonly seen in the younger, throwing athletes. Abrams⁴⁵ describes secondary impingement and the relationship of shoulder instability and rotator cuff impingement. He reports that in the throwing shoulder, repetitive stresses and microtrauma may cause the static stabilizers (labrum and capsular ligaments) to become stretched, resulting in increased translation of the glenohumeral joint. The rotator cuff must exert additional tension to control this translation. Overuse tendinitis may occur as the cuff fatigues from eccentric overload. With cuff fatigue comes a decreased ability to control the humeral head, and anterior-superior migration of the head results, as well as increased scapular protraction. The subacromial space is reduced and secondary mech-

anical impingement occurs as the supraspinatus tendon is stressed against the coracoacromial arch.

Degenerative Causes

From the above discussion of IS etiology, it is evident that there is no single factor determined to be solely responsible for the symptoms and pathology of IS. It is likely that a combination of elements exists. Neviaser and Neviaser³ state that the common denominator in all of the above theories is the role of degeneration. They support this with the facts that most rotator cuff ruptures are seen in middle-aged and older patients and degeneration of the rotator cuff tendons are associated with aging. Bateman⁵ declares that the common foundation of shoulder disorders is a degeneration of the rotator cuff or a tendinitis.

Fu et al⁴⁴ state that both extrinsic and intrinsic factors may be involved with the pathomechanics of rotator cuff tear. Extrinsic causes occur from an outside structure such as the coracoacromial arch. Neer's¹¹ view that 95% of cuff tears are due to mechanical impingement is disputed by others who believe the majority of rotator cuff tears are initiated by an intrinsic degenerative tendinopathy.^{18,25} The authors support this degenerative theory with the fact that there is increasing incidence and severity of cuff tears with age. The presence of partial tears decreases power of the rotator cuff permitting an upward migration of the humeral head which

goes on to cause a secondary impingement. Ogata and Uhthoff¹⁸ acknowledge that a primary impingement between the superolateral corner of the greater tuberosity and the undersurface of the acromion may occur, especially in the presence of the beaked acromion.

In a study to determine whether morphological changes in the CAL were related to impingement, Uhthoff et al⁴⁶ did not find an excessive proliferation of fibrous tissue. They believed the cause of impingement in the absence of bony encroachment or a thickened CAL to be from an expansion in volume of the cuff tendon or subacromial bursa. Recently Nirschl⁴⁷ has described the intrinsic process of angiofibroblastic hyperplasia which occurs as the initial process and develops into rotator cuff calcification, erosion and secondary impingement.

The link between degeneration and vascularity has also been established. Breakdown changes of tendinitis, calcification and rupture of the tendons of the supraspinatus, infraspinatus and intracapsular part of the biceps were observed in the area of avascularity reported by Rathbun and Macnab.³⁴ A connection between shoulder joint contracture and rotator cuff degeneration is also seen to exist. In a study of 2027 subjects, the highest incidence of contracture was seen in patients aged 40-60 years, suggesting that degeneration of the cuff might play some role in limiting range of motion.¹⁶

In stage 1 and 2 of IS, the inflammatory response of tendinitis may spread to the biceps, subacromial bursa and other soft tissue as well as the supraspinatus tendon. Whether the inflammation is due to mechanical/anatomical or degenerative changes is uncertain. It does appear that tendon vascularity and the presence of microtrauma play roles in the etiology also. Impingement, either primary or secondary, can lead to a vicious cycle involving degenerative changes of the acromion, AC joint, CAL, and rotator cuff and biceps tendons. The cycle continues in the presence of increased volume of the soft tissues or decreased available space in the subacromial joint area. In each individual case, each lesion is determined by the etiologic cause which predominates: but in all cases, the net result is degeneration of the tendons.³

CLINICAL EVALUATION

Subjective Evaluation

The purpose of the subjective evaluation is to determine the source of the patient's complaint, the nature of the complaint, and the severity and stage of the condition.⁴⁸ Pain is often the major complaint of a patient presenting with shoulder dysfunction.^{49,50} It is important to note the location and description of the pain. In stage 1 of IS, the pain may be described as a dull ache about the shoulder following strenuous activity. This may progress to discomfort during sport or activity, with the pain affecting performance and interfering with sleep.⁸ In stage 2, the toothache-like discomfort may interfere with activities of daily living (ADL's), be worse at night, and eventually cause an inability to perform the maneuver which caused the IS.⁴²

Specific questions regarding pain need to be raised by the examiner. The presence of referred pain will need to be determined. Nearly all shoulder structures will refer pain into the C5 dermatome.⁵¹ Pain which has spread distally may indicate the condition is worsening, whereas receding pain indicates the problem is not as severe. The presence of an acute inflammation may be indicated when proximal pain moves distally during a particular movement or activity.⁴⁸

Referred pain to the shoulder may also arise from the cervical spine and less commonly from visceral, pleural, diaphragmatic and cardiac pathology.⁵⁰

The patient must be asked whether there was any past or present trauma which may have caused the problem. The duration of the pain is also important to know; whether it is acute or chronic, and how long the pain has been present.

If the patient is an athlete, specific details must be identified regarding the type of sport, position played, whether or not any changes have been made in the biomechanics of the activity, and the relationship of the pain to a specific activity. If the patient is a worker, the nature of the job and the position of the arms during work activities must be ascertained, as well as the relationship of pain to a specific job task.

In addition to questions regarding pain, Yocum⁴⁹ states that handedness is a key element in the initial historical review. Jobe and Bradley⁵⁰ agree, and relate that most athletes will have shoulder dysfunction involving the dominant extremity unless there has been direct trauma affecting the nondominant side. The age of the patient is also a factor to assist in determining the stage of IS and the pathology which may be involved. The 25 to 40-year-old group (stage 2) generally contains the largest number of patients, but age is variable, and athletes, recreational athletes, and laborers in this age group may also be in stage 1 of IS.⁸

A review of the patient's general health should also be included in the subjective evaluation. The patient should be asked if there has been a history of heart disease, high blood pressure, lung or diaphragmatic conditions, and gall-bladder disease. The examiner should also keep in mind the possibility of neoplastic disease such as the Pancoast tumor, or the presence of metastasis.^{49,52}

Objective Evaluation

Screening Exam.--Cyriax⁵³ states that the first stage of the examination of the shoulder is a quick survey from neck to hand. When the lesion is found in the shoulder, that area is examined more thoroughly. If the pain is referred to the shoulder from another area, examination is concentrated there. The screening examination is designed to differentiate a shoulder problem from one in the cervical spine or elbow, but not to pin point a specific lesion or structure.⁴⁸ Range of motion (ROM) at each joint is performed in an active manner through the entire available range with resistance to motion given with the muscle in a shortened position and the joint at the limit of motion.

Neviaser⁵⁴ states that the neck should be examined in every patient presenting with a chief complaint of shoulder pain. Because cervical spine conditions can refer pain to the shoulder, the screening tests should include a physical

examination of the neck and a neurologic evaluation of the upper extremities.

The sternoclavicular and acromioclavicular joints may generate local symptoms rather than referred symptoms and should be palpated to rule out problems from these joints. Quick tests of ROM and strength at the elbow should also be done in addition to those performed at the shoulder.

Bowling et al⁴⁸ note that a specific examination of the shoulder complex is indicated when there is postural abnormality, deformity or wound, abnormal response to active elevation, weakness, pain or limited ROM on resisted shoulder girdle or glenohumeral elevation, or pain that localizes to the shoulder with resisted elbow flexion or extension.

Observation.--The objective evaluation which seeks to find a test movement that reproduces the patient's symptoms, begins with observation. Obvious surgical incisions and deformities should be noted. Posture of the head, spine and upper limb should be observed without clothing hampering the examiner's view. A slouched sitting posture may indicate tightness of anterior thoracic muscles and weakness of posterior muscles.⁵² The presence of an increased thoracic kyphosis can limit shoulder elevation due to the change in resting position of the scapula.⁴⁸

As atrophy is associated with the end stages of IS, the supraspinatus, infraspinatus and deltoid should be closely examined. A prominent scapular spine may be seen if there

is atrophy of either the supraspinatus or infraspinatus.⁵⁰ Any winging of the scapula should be noted. In an athlete or laborer, there may be relative hypertrophy of the dominant extremity. Baseball pitchers with chronic shoulder discomfort typically have a lower position of the dominant shoulder compared to the nondominant side and a forward posturing of the dominant shoulder with more lateral position of the scapula.⁵⁵

Observation should also include inspection of the synchrony of motion, which refers to identical movements of the shoulder girdle complex bilaterally.⁵⁵ Inman⁵⁶ originally described this by stressing that motion occurs in all the joints of the region simultaneously. He summarized that once 30° of abduction or 60° of flexion had been reached, the ratio of humeral to scapular motion is 2 to 1. The quality of movement is best observed by having the patient bilaterally abduct or flex the upper extremities. Any disturbance in this synchrony or rhythm should be documented.

Range of Motion (ROM)--Both active and passive ROM testing is done during the shoulder evaluation. Active movements include elevation, depression, protraction and retraction of the shoulder girdle, and flexion, extension, abduction, horizontal adduction, internal and external rotation of the glenohumeral joint.⁴⁸ Active movements stress both contractile and noncontractile tissues. Range of motion

may be normal, limited, or excessive. The presence of pain during the movement should also be assessed.

A painful arc of motion is a significant finding in patients with IS. Cyriax⁵¹ states that it is a secondary sign indicating the lesion is located in a pinchable position between the acromion and the tuberosities. The painful arc may often occur during active elevation, but Watson⁵⁷ notes that as the arm is lowered from 120° to 70° , maximum tension is developed in the rotator cuff. If this is painful, it is suggestive of tendinitis.

All movements done actively are also done passively to test primarily the noncontractile tissue.⁵² The end feels of joint motion should be noted, with a capsular end feel (stretching leather) the most common at the glenohumeral joint.⁴⁸ A passive overpressure at the end range may be necessary when both active or passive ROM is normal and pain-free.

To get a true picture of glenohumeral motion, the scapula may be stabilized against the thorax during passive movement testing. In addition, Abrams⁴⁵ suggests that rotation measurements be made both with the elbow at the side and at 90° of abduction. When evaluating athletes, particularly throwers, the examiner should be aware of adaptive changes, such as an increase in external rotation and decrease of internal rotation in the dominant extremity. In a study comparing normal subjects to patients with verified insta-

bility and IS, Warner et al³⁰ found that patients with stage 1 and 2 of IS had marked limitation of active internal rotation and cross-chest adduction compared with instability patients.

Passive accessory motion testing should also be performed to assess joint play and associated irritability. Movements at the glenohumeral joint include distraction, and anterior, posterior, and inferior glides.^{48,52} Findings include normal mobility, hypomobility, and hypermobility. Posterior capsule tightness has been associated with patients with IS.^{7,30,55} The involved shoulder should always be compared with the uninvolved side when testing all active, passive and accessory movements.

Strength.--Manual muscle testing (MMT) is done with the joint in mid-range or neutral to eliminate stress on the noncontractile tissue and evaluate the contractile tissue.⁵¹ Pink and Jobe¹⁴ include tests performed for shoulder flexion, extension, abduction, scapular plane abduction (scaption), glenohumeral rotation, and scapular protraction, retraction, elevation, depression, and upward/downward rotation. Cyriax⁵¹ also tests resisted elbow flexion and extension. The examiner should note whether the resisted test is strong or weak and if it elicits pain. Patients with supraspinatus tendinitis from IS may have pain on resisted abduction or external rotation.¹⁰ In stages 3 and 4 of IS,

these tests may be either painless and weak, or painful and weak; depending on the chronicity of the rotator cuff tear.⁴⁸

The supraspinatus or "empty can" test is a very useful test for IS. Through EMG studies, Jobe and Jobe² found this test to isolate the supraspinatus, with the remaining cuff muscles comparatively inactive. They describe first assessing the deltoid with the arm at 90° of abduction and neutral rotation. The examiner next gives resistance with the shoulder internally rotated (thumbs pointing down) and angled forward 30°. The glenohumeral rotators are typically tested with the elbow at the side, but in a racquet or throwing athlete, it is functionally appropriate to also test at a position of 90° abduction.⁵⁸ Also in athletes with overuse injuries from IS, it may be necessary to examine them after the condition has been aggravated by activity, as a MMT may not produce enough stress to reproduce the symptoms.⁴⁸

The scapular rotators cannot be neglected. The serratus anterior plays a key role in throwing and swimming.⁵⁹ Weakness can be shown by having the patient do a "push-up" off the wall and watching for any signs of scapular winging.⁴⁹

Strength deficits and muscle imbalances may not be obvious on MMT, but may be evident with isokinetic testing procedures. Normative data is available. In a study of 30 normal volunteers, Ivey et al⁶⁰ reported isokinetic strength ratios of 3:2 for internal rotation to external rotation, 5:4 for extension to flexion, and 2:1 for adduction

to abduction. Warner et al³⁰ tested 30 shoulders of normal subjects for internal and external rotation and found the ratio to range from 120% to 150% with the dominant side ratio 30% greater than the nondominant side. When doing comparisons however, it is important to realize that testing positions and speeds may vary from study to study.

Neurovascular Assessment.--A neurovascular assessment may be necessary when there is reported paresthesia or anesthesia, a known injury capable of producing neurological insult, or when there is weakness of a muscle during MMT.⁴⁸ This should include testing deep tendon reflexes and sensation in all dermatomes. To rule out thoracic outlet syndrome and other vascular problems, the vascular status of the arm is examined by palpation of the distal arteries with the arm in various positions.⁴⁹

Palpation.--Bowling et al⁴⁸ state that palpation for tenderness should be done after the other parts of the functional examination have been completed. Structures to be palpated include the sternoclavicular joint, AC joint, and clavicle. At the glenohumeral joint, structures include the subdeltoid bursa, tuberosities, rotator cuff tendons, and long head of the biceps tendon. By passively extending the shoulder, direct rotator cuff tendon palpation can be accomplished as the site of insertion is rotated anteriorly out from underneath the acromion.⁶¹ Along with tenderness to palpation of the rotator cuff tendons, the CAL is often

tender in patients with IS, as is the anterior acromion.⁴⁶ Crepitation about the acromion may also be noted.⁵⁰ Hoppenfeld⁶² describes and illustrates specific palpation techniques about the shoulder.

Special tests.--Special tests are useful in the diagnosis of IS. The impingement tests are illustrated in Figure 5. The first test consists of forced elevation of the humerus against the anterior acromion. The scapula may be stabilized when this is done. A physician may also perform this test by injecting 10 ml of 1% Xylocaine beneath the acromion.⁶³ If the pain caused by the forced elevation is eased by the injection, an impingement may be present. Hawkins and Kennedy⁴² describe another method of performing the impingement sign. At 90° of forward flexion, the arm is forcibly internally rotated, jamming the supraspinatus tendon against the anterior surface of the CAL and reproducing the patient's pain. If impingement of the biceps tendon is suspected, internal rotation of the arm in this position will cause a disappearance of the painful catching sensation. The rotation clears the tendon of the biceps long head from the coracoacromial arch.⁵⁸

The "empty can" position for testing strength of the supraspinatus may also effectively recreate impingement.⁶¹ The test is useful for demonstrating weakness of the supraspinatus, possibly indicating rotator cuff tears or the later stages of IS. The drop arm test may also provide evidence

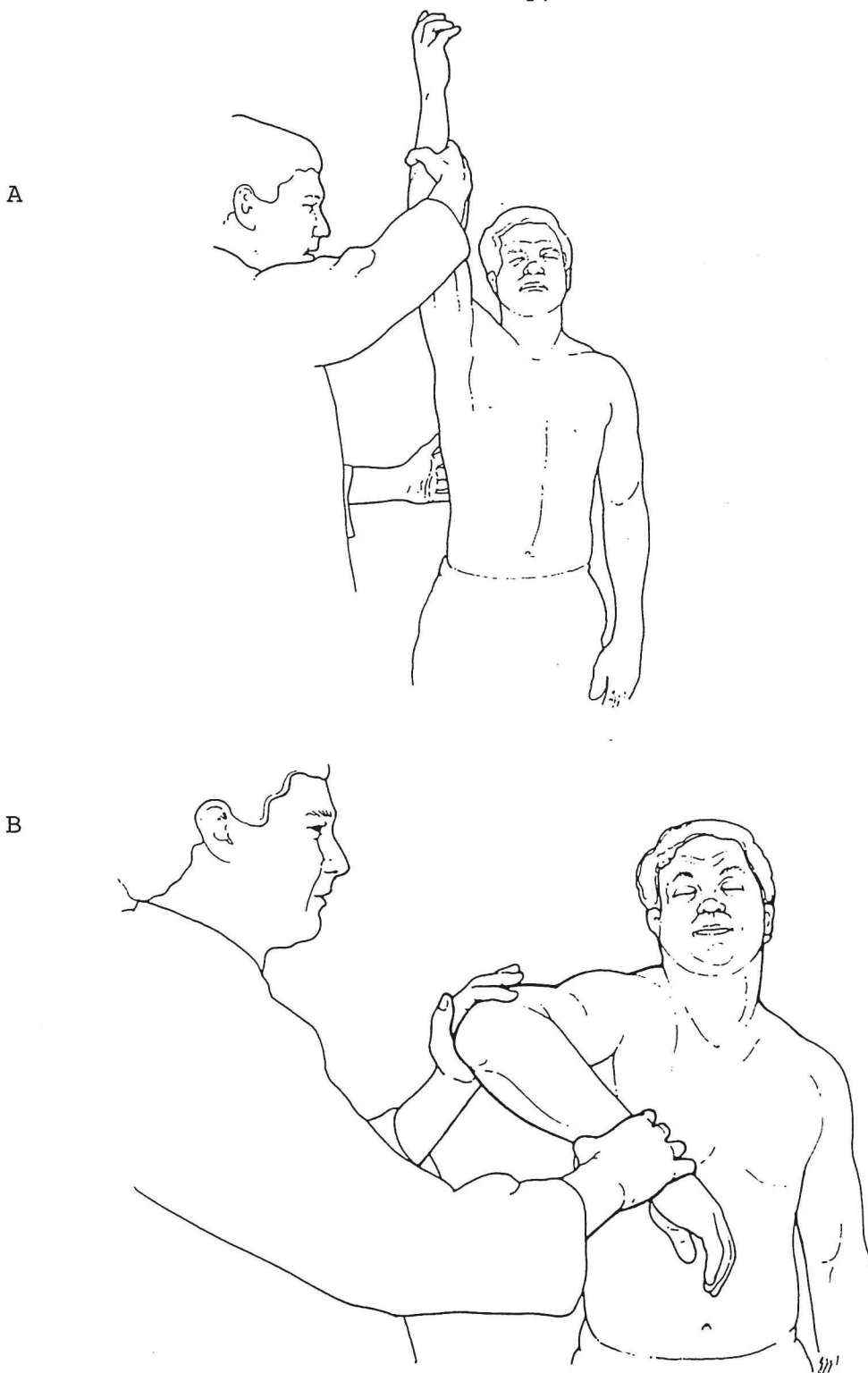


Fig 5.--Tests for impingement. A) Forced elevation of the humerus. B) 90° forward flexion with forced internal rotation. (From Jobe FW, Bradley JP. The diagnosis and nonoperative treatment of shoulder injuries in athletes. Clin Sports Med. 1989;8:425-426.)

for a rotator cuff tear. In this test, the shoulder is abducted to 90° and the patient is then asked to lower the arm slowly to the side in the same arc. A positive finding is severe pain or the inability to lower the arm slowly.⁵⁰ This test is usually not positive in the young athlete, but may be seen in the older population with large degenerative tears.

Since impingement and instability may be part of a continuous process, especially in the young athlete, even subtle signs of instability should be investigated. Jobe and Kvitne⁶⁴ feel the most sensitive method for detection of occult anterior glenohumeral subluxation is the apprehension test followed by the relocation test (Fig 6). With the patient supine, the arm is abducted to 90° and externally rotated while the examiner gently pushes anteriorly on the humeral head. This will generally be uncomfortable for a patient with anterior subluxation. This test is then repeated with a posteriorly directed force on the humeral head. There will be no change in pain level for those with impingement, but the patients with instability will now be able to tolerate maximal external rotation with the humeral head in a reduced position.

Other special tests may be necessary to further evaluate the structures about the shoulder. Because the biceps tendon is adjacent to the undersurface of the supraspinatus tendon, it may also be painful when IS is present. Pain along the

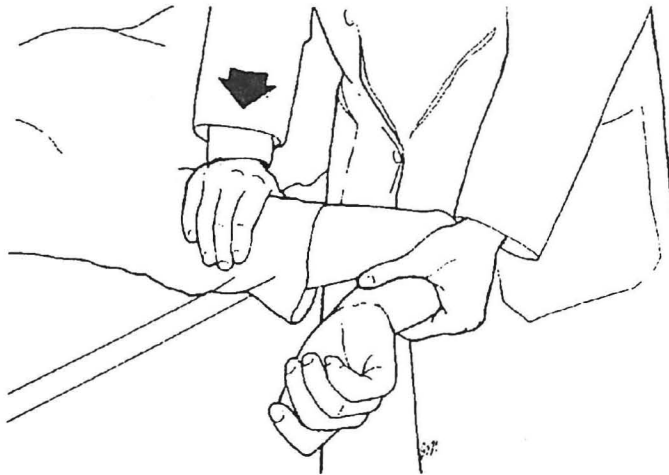
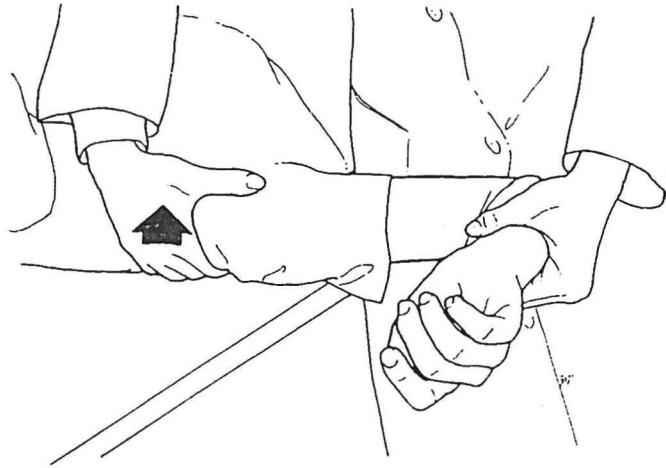


Fig 6.--Anterior instability test followed by relocation test. (From Pink M, Jobe FW. Shoulder injuries in athletes. Clin Manage. November/December 1991;11:p 42.)

bicipital groove is elicited by resisted shoulder flexion with the elbow extended and forearm supinated. This is known as Speed's sign.⁸ Pain in this same region may also be caused by positioning the elbow at the side and flexed to 90°, and resisting supination. This represents a positive Yergason sign.⁸

The quadrant and locking position may reproduce pain in IS patients but should be reserved for those patients who have not responded positively to the other less strenuous tests.⁵² The quadrant position is assessed with the patient supine and the scapula stabilized by the therapist. The patient's arm is then taken into full flexion and external rotation. The degree of abduction is varied to locate a point that reproduces the patient's pain.⁶⁵ The locking position is done with the patient supine, and while the scapula is stabilized, the arm is hyperextended and then abducted to a point where rotation is locked.⁶⁵

Other diagnostic studies can be ordered by physicians to corroborate the physical examination. Roentgenographic signs of impingement seen in the later stages include an anterior acromial spur and subchondral sclerosis or cysts of the greater tuberosity.⁵⁷ Identification of a type III or hooked acromion, would support the diagnosis of IS. In the older recreational athlete, a decrease in the humeral acromial interval (less than 6 mm) along with other bony changes can be indicative of rotator cuff tear.⁵⁰

The double contrast arthrogram is widely used to help diagnose IS as well as other shoulder pathologies.⁵⁰ The CT scan and MRI are less commonly used for diagnosing IS, but may become more useful in the future.

The therapist must be aware of the differential diagnosis involving anterior shoulder pain. These include: shoulder instability, AC joint pathology, cervical root irritation/cervical spondylosis, calcific tendinitis, frozen shoulder, suprascapular nerve injury and neoplasm.^{1,54} Cyriax⁵¹ also lists psychogenic pain as a differential diagnosis for limitation of non-capsular pattern restrictions of passive movements.

TREATMENT

Once the diagnosis of IS has been made, the emphasis on conservative treatment becomes paramount. Stage 1 symptoms are reversible, but may last up to 18 months.¹ Stage 2 symptoms, if not completely reversible, will be decreased through the use of conservative treatment. Watson⁵⁷ states the only contraindication to at least 9 to 12 months of conservative treatment is the presence of a full-thickness rotator cuff tear. For athletes, an extended period of conservative treatment is the best chance they have of returning to high-level competitive activity.

Establishing the correct diagnosis will effect the outcome of conservative treatment. Appropriate treatment depends on an accurate appraisal of the factors and structures which contribute to impingement, so that further musculotendinous damage can be avoided.⁶¹ The overlap of impingement and instability comes into play when exercise programs are prescribed for the overhead throwing athlete. Efforts to improve IS through a conservative or even surgical treatment method may be fruitless if instability is coexistent. In a report of 65 patients who had a failed acromioplasty for IS without rotator cuff tear, 27 shoulders were found to have diagnostic errors, with 7 of those due to instability.⁶⁶

Assuming an accurate diagnosis of IS without rotator cuff tear has been made, treatment goals are based on decreasing rotator cuff inflammation and increasing shoulder strength.⁶⁷ As the inflammation decreases, greater excursion is allowed for the tendons and bursa without impingement. And by increasing rotator cuff and scapular muscle strength to allow better depression of the humeral head, the available subacromial space is maximized.

The phases of treatment to address these goals will be divided into the initial phase, the rehabilitative phase and prevention. Tables 1 and 2 summarize goals and treatment methods for the initial and rehabilitation phase. A key role in prevention and treatment phases will be played by the physical therapist to develop an appropriate exercise and preventative program and assist the patient in returning to their prior functioning status.

Initial Phase

Medical Management.--Although patients with IS may not have visibly evident edema or easy to detect tissue temperature elevation, inflammatory responses have occurred and must be addressed in the initial phase of treatment.⁶⁸ Physicians may prescribe nonsteroidal anti-inflammatory drugs (NSAID's). Two or three steroid injections may be given, but in stage 1, Neer and Welsh⁶³ warn that hydrocortone

Table 1.--Initial Treatment Phase - Goals and Methods

Goals	Methods
Decrease inflammation and decrease pain	NSAID's 1-3 steroid injections Cryotherapy Superficial heat in subacute phase Electrotherapeutic agents Massage Rest Sport or job modification

Table 2.--Rehabilitation Treatment Phase -
Goals and Methods

Goals	Methods
Obtain normal range of motion	Joint Mobilization Stretching Passive range of motion PNF techniques Modalities if needed Post. capsule stretching
Increase strength	Synchrony of motion Isometrics Isotonics Use of equipment Isokinetics Isofunctional exercise

injections may potentially damage the rotator cuff tendons and should be avoided.

Modalities.--Physical therapy modalities are beneficial in decreasing inflammation. Perhaps the most widely used agent is ice. Following acute injury, ice application in 10 minute intervals for the first 24 to 48 hours is the modality of choice to decrease inflammation and pain.^{67,69} Cold packs, ice massage, or ice towels in the acute phase serve to restrict hemorrhage and edema and reduce the metabolic demands of the injured tissue.⁷⁰

In the subacute phase, superficial heat to hasten tissue repair may be beneficial. Rocks⁷⁰ reports that mild heating will not increase the inflammatory process and will relieve ischemic pain. Deeper heating agents such as diathermy or ultrasound have been used to treat IS and other shoulder pathologies, but results may be questionable. In a double blind study of 20 patients with a diagnosis of subacromial bursitis, ultrasound was found to have no further benefit over treatment with ROM and NSAID's or ROM exercise.⁷¹ In many cases of chronic inflammatory conditions, ultrasound may exacerbate the symptoms of swelling, pain, and disability and should be avoided.^{8,39}

Electrotherapeutic modalities can be an adjunct to treatment in the acute or chronic phase of IS. Richardson³⁹ has reported good clinical results using transcutaneous nerve stimulation (TENS) or electrogalvanic stimulation (EGS) to

treat overuse syndromes about the shoulder. The use of TENS was also noted to be clinically successful in managing chronic shoulder pain of swimmers who had been resistant to other forms of conservative therapy.⁴² Wallace⁷² reports that microamperage electrical nerve stimulation (MENS) is clinically successful in decreasing pain caused by IS up to 50% after the first visit.

Massage techniques may also be useful in treating IS pain and inflammation. Friction massage is advocated by Cyriax⁵³ who places the patient's arm in specific positions to isolate the tendons of the rotator cuff. The massage is applied transversely to the tendons to mobilize the connective tissue in patients where irritability is not acute. It may be done for 2 to 20 minutes, depending on patient tolerance, to the spot of maximum tenderness.⁵² Based on rotator cuff vascularity, Erb⁷³ believes a lubricated effleurage massage, done proximal to distal, to the supraspinatus and infraspinatus muscles with the arm abducted 45°, will assist healing and circulation of the tissues. As part of a specific protocol, the massage may be done at any stage of impingement.

Rest.--Rest, in the initial phase of IS treatment, is appropriate for both athletes and nonathletes. According to Moynes,⁶⁹ stopping the overhead activity responsible for producing symptoms, including swimming, throwing, or serving, must be done. In one specific tennis treatment protocol,

the athlete is not allowed to play tennis for 10 days to 2 weeks.¹² An individual who does overhead work should also stop that specific symptom producing activity.

Rest does not mean the patient should be placed in a sling, as this would lead to further rotator cuff weakness.⁵⁰ Moynes⁶⁹ also states that during the period of rest, it is important that the rotator cuff not become contracted from the effects of immobilization.

Modification of a particular sport or job duty may be indicated to allow rest, yet still enable the patient to participate in certain aspects of the sport or job. For example, tennis players may need to avoid the serve and overhead smash shot, and pitchers could switch to a side-arm throw to selectively rest the involved shoulder muscles.⁸ Swimming style may also be modified to prevent excessive shoulder flexion and internal rotation which will exacerbate IS.⁷⁴ Reducing mileage swam, and repetition of throws and serves are also modifications that can be made if the athlete is unwilling to rest completely from the overhead activity.

For workers involved in overhead work, modification of their job may mean light-duty activities or a rotation to a different task which would not require overhead work or lifting.

While a patient is selectively resting the shoulder, it is important to maintain aerobic capability. A worker may use a stationary bicycle or walk for cardiovascular fit-

ness, and use weights for lower extremity strengthening and conditioning. An athlete can concentrate on the uninvolved upper extremity strength as well as aerobic conditioning and strength training using the lower extremities. Working on other aspects of the sport which do not involve the shoulders, such as kicking for a swimmer and leg work for a tennis player, will help keep the athlete's interest and motivation in the sport.³⁹

Rehabilitation Phase

Mobility.--An important goal of the rehabilitation phase is to restore normal glenohumeral motion. Pappas et al⁵⁵ state that it is an absolute necessity to achieve normal glenohumeral and scapulothoracic motion before strengthening programs are begun. This premise was made in regards to the pitching shoulder, but can certainly be expanded to include other sports and patient populations. Without adequate ROM, abnormal movement patterns may be produced. It is best advised to obtain this mobility in a pain-free manner so as not to increase inflammation by being too aggressive.⁵²

Glenohumeral joint mobilization is one effective method of improving ROM without increasing pain or inflammation. Mobilizations are begun in a comfortable and neutral shoulder position with small amplitude movements. Progression may be made to large and small amplitudes at or near the end

available range of motion.⁵⁸ These movements include: caudal glide to increase shoulder elevation, anterior glide to increase external rotation, posterior glide to increase internal rotation and flexion, and lateral glide or distraction to facilitate all glenohumeral movements.^{58,61,68} In the case of anterior instability, the use of anterior glide should be avoided to prevent further damage to the anterior capsule. Scapulothoracic mobilization may also be helpful if restricted scapular movement is present.

To prevent healing of the rotator cuff from occurring in a contracted position, stretching and passive range of motion exercises should accompany joint mobilization. Jobe and Moynes¹³ suggest stretching into external rotation in a supine position with the shoulder abducted to 90° and elbow flexed 90°. An additional stretch is done in supine with external rotation, but with the shoulder elevated 135° and the elbow extended.

This type of stretching should be avoided, however, in throwers suspected of having subtle anterior instability.⁴⁵ In these athletes, posterior capsule tightness should be eliminated to lessen anterior subluxation and the chance of impingement.⁷⁵ Figure 7 demonstrates stretching for internal rotation, flexion, and cross-chest adduction which should be emphasized to increase ROM and stretch the posterior capsule. As part of a total program, Abrams⁴⁵ also suggests stretching the low back and hamstrings.

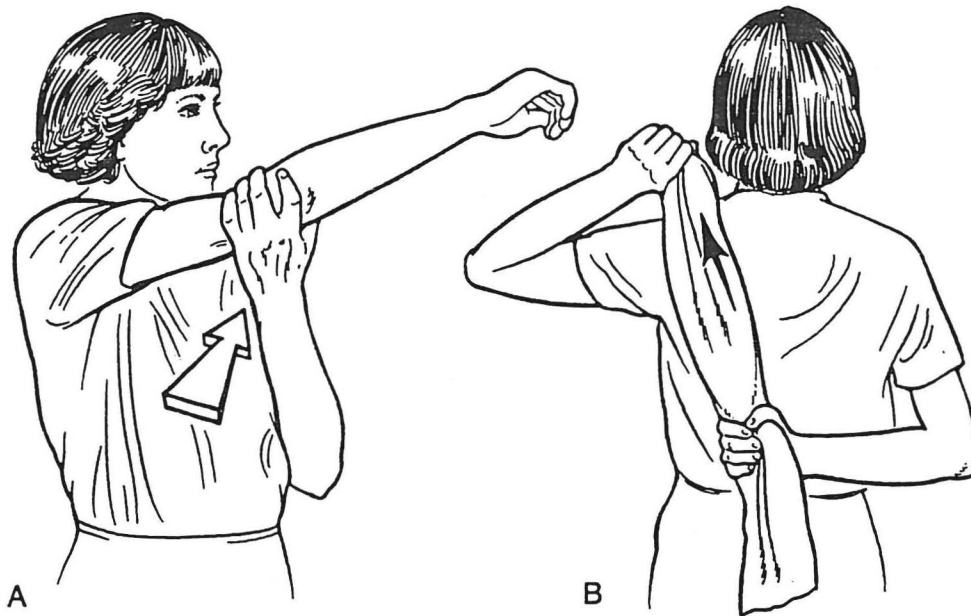


Fig 7.--Posterior capsule stretching. A) Cross chest adduction. B) Internal rotation. (From Matsen FA, Arntz CT. Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The Shoulder. Philadelphia, PA: WB Saunders Co; 1990:p 624.)

Diagonal patterns of stretching such as done in proprioceptive neuromuscular facilitation (PNF) techniques will also help stretch the glenohumeral joint and shoulder girdle complex. Hold relax, contract relax, and slow reversal hold methods of PNF have been found to be very effective in treating the athlete.⁷⁶ All stretching should be done within patient tolerance and should not increase pain. Whatever the stretching technique, the therapist should instruct the patient to perform active motion into the newly gained ROM after the passive stretching has been completed.

Isometric Exercise.--As ROM is increased and pain and inflammation is decreased, strengthening exercises may be initiated. Isometric exercises have the advantage of enhancing muscle strength without joint motion and are necessary when dynamic activities are too painful.⁶¹ Isometric exercise for internal and external rotation done at multiple angles in a pain-free ROM are initially performed.⁵⁸ The therapist can provide resistance manually and a home program can also be given with the patient using a wall, door jam, or uninvolved extremity as the stationary object of resistance. It is recommended that the isometric exercise also include the other shoulder groups, the biceps, triceps, and scapular stabilizers, and be done in a pain-free manner. Exercises may be done 10-20 times each, 3 times a day.⁶⁷

During the early stages of rehabilitation, it is important to continue with the cardiovascular program. Also at

this point, the therapist can initiate exercise to restore synchrony of motion. Sequencing symmetrical scapulohumeral rhythm would include depression of the humeral head followed by external rotation and abduction done in a pain-free ROM. Pappas et al⁵⁵ recommend teaching the sequence of active abduction followed by horizontal extension and external rotation, which is the required normal pattern for the throwing athlete in the cocking phase. The therapist can begin with passive movements in these patterns, gradually adding resistance as the patient tolerates and as the movements become more synchronous and symmetrical. Good visual cues are provided by mirrors in addition to the tactile cues of the therapist.

Isotonic Exercise.--The patient may be advanced to isotonic exercise as tolerance for exercise and strength increases. Strengthening of the rotator cuff muscles is necessary as these dynamic muscles center the humeral head in the glenoid to provide stability. Strengthening the scapular rotators contributes to restoring normal scapulothoracic rhythm and positioning the scapula for stability during overhead activity.⁵⁹

Strengthening of these muscle groups should be done in a pain-free manner avoiding ranges of motion which could exacerbate the impingement. Of the four rotator cuff muscles, the supraspinatus is emphasized for its role in stability and is selectively strengthened. Ellenbecker and Derscheid⁵⁸

suggest the empty can position for strengthening the supraspinatus beginning in supine (partially gravity eliminated), and progressing to upright postures (anti-gravity) as tolerated. To prevent further impingement, a range of 0° to less than 90° of motion is suggested. Internal and external rotation exercises are used for the remaining cuff muscles with resistance gradually progressed according to the patient's response.

In addition to the rotator cuff muscles, the muscles which position the humerus must also be strengthened.¹⁴ These include the three heads of the deltoid, the pectoralis major and the latissimus dorsi. An EMG study was undertaken by Townsend et al⁷⁷ to determine a core group of exercises to be included in a rehabilitation program for the throwing athlete. The rotator cuff and humeral positioners were studied by analyzing seventeen specific exercises. Three exercises were consistently among the top two for every muscle tested. Scapular plane elevation (scaption) in internal rotation was the best exercise for the anterior and middle deltoids and subscapularis, and second best for the supraspinatus (Fig 8). Prone horizontal abduction with external rotation had the highest EMG activity for the infraspinatus and second highest for the teres minor and posterior deltoid. The seated press-up was the top exercise for the latissimus dorsi and pectoralis major. Although not selected as a core exercise, the seated military press was the best exercise

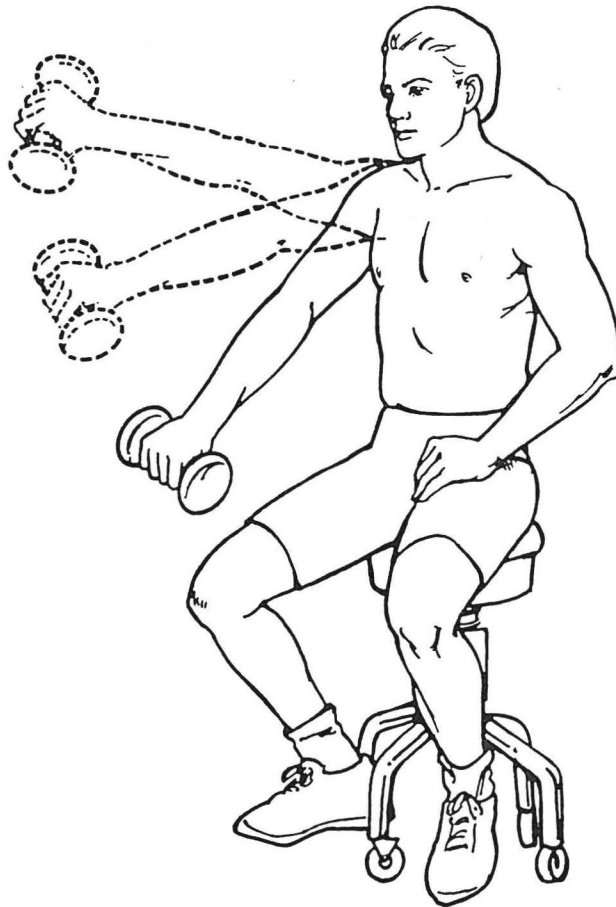


Fig 8.--Scapular plane elevation (scaption) in internal rotation. (From Townsend H, Jobe FW, Pink M, Perry J. Electromyographic analysis of the glenohumeral muscles during a baseball rehabilitation program. Am J Sports Med. 1991;19: p 265.)

for supraspinatus activity. The authors noted that forward flexion may be a more ideal exercise than scaption as the muscle activity patterns are similar but the risk of impingement is lower.

The scapular rotators cannot be overlooked in a conservative treatment program for IS. These muscles include the trapezius (all portions), levator scapulae, rhomboid, pectoralis minor and serratus anterior. Resistive exercises for these muscle groups help restore a stable scapular base. In swimmers, for example, an EMG study done on dry land indicated the serratus anterior works at near maximal level during the recovery phase.⁷⁸ The authors concluded that a vigorous strengthening program for the serratus anterior and scapular rotators could be an important factor in alleviating impingement.

The scapular rotators may be strengthened by using light weights or other resistance for the motions of scapular retraction, protraction, elevation, and depression. An EMG study done by Moseley et al⁷⁹ examined 16 exercises used in a shoulder rehabilitation program. EMG activity of the 8 scapular rotators was analyzed. Scaption with external rotation was found to be an optimal exercise. Prone rowing was selected for its range of scapular retraction and peak muscle activity. A push-up with a plus (end range protraction) was found beneficial for protraction produced by the serratus anterior and pectoralis minor. The last core exer-

cise selected was the seated press-up which was the best exercise for the pectoralis minor.

These exercises should be incorporated in the core program, however, the therapist will need to select other exercises to augment this basic program and be patient specific. The diagonal movement patterns incorporated in PNF techniques are also useful for strengthening a combination of muscles in more functional patterns. Distal upper extremity muscles must not be neglected.

The use of exercise equipment is another way to augment a basic exercise program when the patient can tolerate manual resistance exercises without pain. Free weights may be used, beginning with low weight of 1-2 pounds and increasing to 5 pounds.⁴⁵ Resistive elastic bands or surgical tubing can be used to strengthen most muscle groups and have proven to be effective. The effect of Theraband (Hygenic Corporation, Akron, OH) on strength of the internal rotators was studied in 28 subjects.⁸⁰ A 10% strength gain was demonstrated in the experimental group compared to the control group, with the greatest gains occurring eccentrically.

Internal and external rotation strengthening exercises can be done with the assistance of pulley mechanisms. Both eccentric and concentric contractions are accomplished and pulleys are thought to provide a more uniform resistance through the entire range of motion.⁵² Wall pulleys can also

be designed and set up to strengthen the other glenohumeral and scapular muscles.

The Upper Body Ergometer (UBE, Lumex Inc., Ronkonkoma, NY) and Schwinn Aerodyne (Schwinn, Chicago, IL) are recommended for strengthening scapular stabilizers, training the cardiovascular system, and increasing upper extremity muscle endurance.⁵⁸ More sophisticated equipment available on the market includes variable resistance isotonic strengthening systems. The specific pieces of equipment used, the amount of resistance, and the range of motion the exercise is performed through, should be determined by the therapist's assessment of each individual patient's needs and symptoms. Any use of equipment should be closely monitored to prevent exacerbation of IS symptoms.

Position for Exercise.--Controversy exists over what position the shoulder should be in during strengthening exercises. There are therapists and physicians who advocate that rotator cuff exercises for rotation be done with the arm held close to the body.^{13,14,69} Other sources cite the "wringing out" theory of the supraspinatus tendon and recommend that the exercises be done with the arm abducted from the side of the body.^{58,61} A position of slight abduction and forward flexion has been suggested.⁵⁸ To achieve this modified position, a pillow or towel roll can be placed between the patient's side and the exercising extremity. The arm may also be supported out to the side slightly by

the contralateral arm when doing surgical tubing or elastic band exercise. For patient comfort, Warner⁸¹ notes that an intermediate zone of abduction is best when doing rotation exercises.

Is the adducted position better than the mid-range position when muscle activity of the rotators is considered? Belle and Hawkins⁸² studied EMG data of the supraspinatus, infraspinatus, and subscapularis muscles during external rotation strengthening exercise with the arm in neutral and in 40° of abduction. They concluded that rotational exercises do strengthen the humeral rotators, but there is little difference in muscle activity between the two positions.

For isotonic strengthening of abduction and flexion, a range of 15° to 75° should be adhered to initially, avoiding the horizontal position.⁶¹ Care must also be taken to avoid positions that would stress the anterior capsule in a patient suspected of having instability. For example, to prevent anterior head displacement during a push-up, the patient should be cautioned to not lower the body completely to the wall or floor.¹⁴

Principles of Exercise.--Exercises should emphasize low resistance and high repetitions in a pain-free range of motion. Pappas et al⁵⁵ find there is no need to exceed 5 pounds of resistance when rehabilitating the pitching shoulder, as weights larger than this tend to precipitate a loss of muscular balance around the shoulder.

Another current concept in rehabilitation of overhead athletes is the dysfunction in the rotator cuff caused by fiber failure and the loss of eccentric control.⁷⁵ The balance of eccentric and concentric strengthening is especially important in the throwing sports that involve a follow-through phase. Deceleration of the arm in this phase requires eccentric contractions and adequate endurance to prevent overload.⁵⁵ Whether using manual resistance, free weights, pulleys, or resistive bands, the patient must be taught to return the extremity slowly to the starting position while under load. Multiple sets at low resistances done both concentrically and eccentrically will help achieve strengthening goals.

Isokinetic Exercise.--Although not available in all clinics, isokinetic equipment is an additional form of resistance for strengthening the rotator cuff and shoulder groups. It is expensive and time consuming to set up when compared to other forms of exercise, but is beneficial in that it provides accommodating resistance through a range of motion at preselected speeds. Athletes will especially benefit from the higher speed capabilities afforded through isokinetic exercise as the upper extremity involved in many sports must function at high speeds.

Ellenbecker and Derscheid⁵⁸ recommend beginning with internal and external rotation submaximal contractions done in a pain-free range of motion. Caudal glide and humeral

head depression can be facilitated by initially concentrating on rotation exercise (isotonic or isokinetic). Velocities ranging from 180 to 300 degrees per second ($^{\circ}$ /sec) are felt most appropriate for patients with IS.^{58,61} Performing these exercises with the arm positioned in slight abduction and flexion is preferable to the 90° abducted position, and can be accomplished by tilting the dynamometer head back 20° to 30° from the horizontal.⁵⁸ Additional range of motion stops can also be placed to limit the amount of rotation performed in each direction in the case of pain or instability.

Progression is possible if there are no increased symptoms with the initial protocol. The patient can advance toward maximal intensity contractions and increased number of sets in internal and external rotation. Other shoulder motions of flexion/extension and abduction/adduction are also incorporated to promote the remaining force couple interaction. Abrams⁴⁵ suggests initially blocking flexion at 90° and abduction at 60° in patients with a primary diagnosis of IS. Along with this phase of treatment, it may be necessary to continue with modalities, joint mobilization, stretching, cardiovascular training, and a home strengthening program, depending on the needs of the patient.

One significant advantage of isokinetic systems is the ability to generate objective, numerical data regarding a patients' strength, power, and endurance. A baseline can

be established, with subsequent tests done to determine progress toward goals. In comparing patient data to established norms, it is imperative to understand the differences possible due to equipment, patient set-up, test speed, and protocol.

As stated previously, normal ratios for the shoulder antagonists have been published.^{30,60} The therapist also needs to consider the factor of extremity dominance when comparing ratios and other data. For internal to external rotation, Warner et al³⁰ reported a 30% higher ratio on the dominant side at speeds of 90 and 180 degrees per second when tested in the modified abducted position. It would not be sufficient then, for an athlete to have the dominant, involved extremity rehabilitated to just 100% of the nondominant uninvolved extremity. With their data indicating a weakness of external rotation in patients with IS, a therapist must pay close attention to the external rotators and emphasize this in the isokinetic program and throughout the rehabilitative phase.

In another study, Cook et al⁸³ compared ratios of flexion/extension and external/internal rotation at speeds of 180 and 300°/sec, tested in supine, in pitchers and non-pitchers. They reported a weakness of external rotation strength and increase in internal rotation strength in the throwing arm compared to the nonthrowing arm of pitchers, especially at the speed of 180°/sec. No significant difference was noted for flexion/extension between pitchers' arms.

The authors believed that clinically, to reduce risk of shoulder injury, attention must be paid to maintaining or strengthening the external rotators of pitchers. A similar result of relative decrease in external rotation strength in the dominant arms of professional pitchers was reported by Ellenbecker et al.⁸⁴

Isokinetic strengthening is now possible in either concentric or eccentric modes. A study was performed to discover which method would most affect functional performance.⁸⁵ Ball velocity during a tennis serve was the functional performance measurement tested in 22 college varsity tennis players. Results from the study demonstrated gains in power and muscle explosiveness for the shoulder rotators for both concentric and eccentric testing methods. The concentric training group showed a significant improvement in speed of tennis serve compared to the eccentric group. In addition, the authors concluded that strengthening the internal and external rotators was of great importance in preventive conditioning and rehabilitation of patients with shoulder overuse injuries.

Return to Activity.--When the athlete is pain-free and able to perform the previous patterns involved in their sport, they are able to begin isofunctional exercise. These exercises are sport and muscle specific.⁷⁶ Lehman¹² describes exercises using the tennis racquet and resistive bands to selectively strengthen muscle groups. Other authors have delineated a return to pitching program beginning with mirror

throwing and progressing to short distance and long distance arc throwing.⁵⁵

For athletes, the final step in rehabilitation of shoulder IS is the correction or modification of the abnormal mechanics and patterns of use which contributed to the original pathology. The therapist can videotape the athlete, or if available, the athlete may be directed to a biomechanical analysis expert or high-level coach to work on correction of the abnormal movements or patterns.⁵⁸

For a patient returning to work, modifications may be necessary in the work place, and the patient should receive education in body mechanics and ergonomics. The act of repetitive overhead lifting is modified through lightening the load, changing to a long-handled tool, or raising the body on a ladder or platform in order to use two hands and work below shoulder level.⁵ The therapist can place the patient in a work hardening program to promote the strength and endurance needed for the patient to return to their job. Job site analysis is also beneficial for the patient involved in overhead job tasks.

Prevention

With shoulder IS being such a common problem, it would be wise to develop a preventative program directed at athletes and workers who would be susceptible to the pathology.

Athletes should use warm-up exercises prior to every workout. This serves to increase tissue temperature and may consist of general body warming, such as slow jogging, and also sport specific and flexibility exercises.⁶⁹ Stretching exercises may incorporate static stretches, however, the PNF techniques, including slow reversal hold, are noted to be very successfully used for athletes.⁷⁶

Stretching of the posterior shoulder capsule should also be incorporated into the preventative program. The cross-chest adduction stretch and internal rotation stretch should be taught to all athletes, especially those involved in throwing or swimming. Workers who perform overhead duties can easily do these stretches before their shift starts or at breaks.

A preventative program would also include strengthening exercises. For the athlete, a strength evaluation would determine specific muscles to be focused on. For throwers, emphasis on the external rotators of the dominant arm may decrease the risk of shoulder injury.⁸³ For swimmers, the serratus anterior and other scapular rotators have been identified as key muscles to strengthen to prevent impingement.⁷⁸ If the athlete is instructed in a strengthening program to address the specific needs of their sport, as well as a good conditioning and flexibility program, they will be at better odds for reducing their risk of shoulder injury and IS.

Free weights, or the use of equipment such as pulleys, or resistive bands, are perhaps the less complex of the methods which can be used for preventative strengthening. The therapist must be sure to stress proper technique and form in order to prevent the athlete from working in the zones where impingement may occur. Education will also help alert the athlete to signs or symptoms of IS so that early treatment interventions may be taken.

Isokinetic equipment is another method of strengthening which is useful as a preventative measure to develop strength and endurance of athletes requiring repetitive use of their shoulders. Janda and Loubert⁸⁶ implemented another method using the techniques of PNF as a preventative program for throwing athletes. It focuses on the neck, upper trunk, and upper extremities. The athletes are initially instructed in the patterns by a trainer, and then continue on their own.

The working population needs proper muscle balance about the shoulder complex as well as the athlete. A strengthening program incorporating exercises for the rotator cuff, glenohumeral and scapular muscles would be good prophylaxis against shoulder pain and impingement. The addition of trunk and lower extremity strengthening exercises assists with improving the worker's overall fitness level and ability to perform manual tasks.

Treatment and prevention is aimed at the individual as they function during the day. But sleeping postures at night must also be considered, as night pain may commonly be experienced during stage 2 of IS. If taking into account the "wringing out" theory, sleep positions of side lying, side lying on the opposite shoulder with the arm across the chest, and supine lying with the arm slightly extended at the shoulder would contribute to this.⁷⁴ Impingement may occur when sleeping prone with the arm above the head. Positions recommended are; supine with the involved elbow supported by a pillow so the arm is held in slight forward flexion and internal rotation, side lying with a pillow under the shoulder and a second pillow staggered slightly for the head, and side lying on the opposite shoulder and cuddling a pillow under the affected arm so it is totally supported.

Surgical Management

With prevention and an early treatment program, conservative management may produce good results in up to 95% of people with stage 1 and 2 of IS.¹⁴ In individuals who do not respond favorably to a conservative program, it may be necessary to consider a surgical approach. In 1972, Neer⁴ described a technique of anterior acromioplasty for decompression of the subacromial area. This procedure has been widely used since that time. One retrospective study was done to determine the success rate of the surgery when all rotator

cuff tendons were intact.⁸⁷ Of 108 patients who had prior conservative treatment for at least 1 year, the success rate for anterior acromioplasty followed by rehabilitation was 87%. A lower success rate has been reported in patients receiving worker's compensation benefits.^{66,87}

Arthroscopic treatment for shoulder IS has been used recently, but severe shoulder weakness, instability, and previously failed surgery are contraindications to this form of subacromial decompression.⁸⁸ The success rates for arthroscopic procedures are similar to open operations, however, there is less morbidity, and a quicker recovery time.

CONCLUSION

The shoulder is a complex joint owing to the four joints which comprise it, the great amount of mobility allowed, and the potential for overuse, instability and impingement. The glenohumeral joint must move in proper synchrony with the other joints of the shoulder complex to allow normal range of motion. The rotator cuff muscles must work effectively with the deltoid and scapular rotators to provide the force couple necessary to elevate the arm in either flexion or abduction. The supraspinatus functions to stabilize the glenohumeral joint as the arm is moved into an elevated position.

Impingement occurs when there is a decrease in the available space of the subacromial region for the rotator cuff tendons, and at times the long head of the biceps tendon, or there is an increase in volume of the subacromial tissues. Impingement will result clinically in pain in the anterior deltoid area with arm elevation between 60 and 120°, pain with resisted testing of abduction and rotation, and pain frequently occurring at night. Impingement syndrome in stage 1 consists of edema and hemorrhage and may progress to stage 2, which is characterized by thickening and fibrosis of the subacromial tissues.

The etiology of impingement syndrome is multi-faceted and perhaps interwoven. Impingement may result from abnormal anatomical or biomechanical variations. The morphology of the acromion, CAL, and AC joints is mainly implicated. Biomechanical causes include weakness of the rotator cuff muscles resulting in proximal humeral head migration and impingement. Instability may also cause a secondary impingement.

Some researchers base the vascular theory of impingement syndrome on cadaveric research which shows the supraspinatus tendon to be virtually hypovascular near its point of insertion. More recent in vivo studies are examining the rotator cuff vascularity but have not supported the research reporting a hypovascular zone.

Degeneration of the tendons of the rotator cuff is also a popular theory of IS, and certainly is a significant factor in the later stages of IS when there is actual tear of the rotator cuff. Whether or not the initial process of impingement is caused by intrinsic factors such as tendon degeneration is unknown.

Microtrauma resulting from shoulder overuse is often an etiologic factor in IS in the young and middle-age athlete as well as the overhead worker. A current concept in working with athletes with IS is the interplay of instability and impingement. The possibility of anterior, posterior, or multi-axial instability should be ruled out whenever a patient is diagnosed with primary impingement.

The diagnosis of IS can be made through a purely clinical evaluation. Special tests such as the impingement sign are very useful to the skilled clinician. The x-ray and arthrogram may also be necessary to verify the diagnosis and rule out rotator cuff defects.

Physical therapy is an important part of nonoperative management of IS. Stages 1 and 2 will often be reversed with appropriate treatment and good follow-through and prevention by the patient. Physical modalities are commonly used in the initial phases of treatment and if needed, are carried over into the rehabilitation phase as well. Rest, activity or sport modification, and the use of NSAID's will also help decrease inflammation of the tendons and bursa and decrease pain.

In the rehabilitation phase, range of motion and exercise in a pain-free manner is emphasized. Obtaining normal range of motion and stretching the posterior shoulder capsule are included as goals of this phase. Exercise can advance from isometrics to isotonics and isokinetics once these goals are achieved. The therapist must prescribe and monitor the exercise program to insure that the patient increases strength of the appropriate glenohumeral and rotator cuff muscles and does not neglect the scapular rotators. Strength of the external rotators must receive special attention. The force couple should be rebalanced to normalize the scapulohumeral rhythm.

Rotational exercises, which are a key part of successful rehabilitation, can be done either isototonically or isometrically. Considering the rotator cuff vascularity, previous EMG studies and patient comfort, these exercises may be more effectively done with the arm slightly flexed and abducted rather than adducted to the side of the body.

The majority of patients with stage 1 and 2 of IS can be successfully rehabilitated to return to their sport or occupation and previous activity level. Modifications in the overhead task may be necessary and prevention of further impingement must be stressed. Once the correct diagnosis of stage 1 or 2 IS has been made, treatment is started immediately and progressed as the patient tolerates.

The physical therapist plays an important role in the evaluation, treatment, and reevaluation of patients with IS. In cases which do not respond to a lengthy period of conservative management, surgery may be necessary.

REFERENCES

- 1 Butters KP, Rockwood CA. Office evaluation and management of the shoulder impingement syndrome. Orthop Clin North Am. 1988;19:755-765.
- 2 Jobe FW, Jobe CM. Painful athletic injuries of the shoulder. Clin Orthop. 1983;173:117-124.
- 3 Neviasser RJ, Neviasser TJ. Observations on impingement. Clin Orthop. 1990;254:60-63.
- 4 Neer CS. Anterior acromioplasty for the chronic impingement syndrome in the shoulder - a preliminary report. J Bone Joint Surg. 1972;54A:41-50.
- 5 Bateman JE. The Shoulder and Neck. 2nd ed. Philadelphia, Pa: WB Saunders Co; 1978:chap 8.
- 6 Depalma AF. Surgery of the Shoulder. 3rd ed. Philadelphia, Pa: JB Lippincott Co; 1983:245-250.
- 7 Matsen FA, Arntz CT. Subacromial impingement. In: Rockwood CA, Matsen FA, eds. The Shoulder. Philadelphia, Pa: WB Saunders Co; 1990:chap 15.
- 8 Hawkins RJ, Abrams JS. Impingement syndrome in the absence of rotator cuff tear (stages 1 and 2). Orthop Clin North Am. 1987;18:373-382.
- 9 Hawkins RJ, Hobeika PE. Impingement syndrome in the athletic shoulder. Clin Sports Med. 1983;2:391-405.
- 10 Matsen FA, Kirby RM. Office evaluation and management of shoulder pain. Orthop Clin North Am. 1982;13:453-475.
- 11 Neer CS. Impingement lesions. Clin Orthop. 1983;173:70-77.
- 12 Lehman RC. Shoulder pain in the competitive tennis player. Clin Sports Med. 1988;7:309-327.
- 13 Jobe FW, Moynes DR. Delineation of diagnostic criteria and a rehabilitation program for rotator cuff injuries. Am J Sports Med. 1982;10:336-339.

- 14 Pink M, Jobe FW. Shoulder injuries in athletes. Clin Manage. November/December 1991;11:39-47.
- 15 Peat M. Functional anatomy of the shoulder complex. Phys Ther. 1986;66:1855-1865.
- 16 Nobuhara K, Sugiyama D, Ikeda H, Makiura M. Contracture of the shoulder. Clin Orthop. 1990;254:105-110.
- 17 Crafts RC. A Textbook of Human Anatomy. New York, NY: John Wiley & Sons; 1966:651-653.
- 18 Ogata S, Uhthoff HK. Acromial enthesopathy and rotator cuff tear - a radiologic postmortem investigation of the coracoacromial arch. Clin Orthop. 1990;254:39-48.
- 19 Perry J. Anatomy and biomechanics of the shoulder in throwing, swimming, gymnastics, and tennis. Clin Sports Med. 1983;2:247-269.
- 20 Rowe CR. Tendinitis, bursitis, impingement, "snapping" scapula, and calcific tendinitis. In: Rowe CR, ed. The Shoulder. New York, NY: Churchill Livingstone; 1988:105-112.
- 21 Brunet ME, Hadad RJ, Porche EB. Rotator cuff impingement syndrome in sports. Physician Sports Med. December 1982;10:86-94.
- 22 Bechtol CO. Biomechanics of the shoulder. Clin Orthop. 1980;146:37-41.
- 23 Patte D. The subcoracoid impingement. Clin Orthop. 1990;254:55-59.
- 24 Penny JN, Welsh RP. Shoulder impingement syndrome in athletes and their surgical management. Am J Sports Med. 1981;9:11-15.
- 25 Ozaki J, Fujimoto S, Nakagawa Y, Mashuhara K, Tamai S. Tears of the rotator cuff of the shoulder associated with pathologic changes in the acromion - a study in cadavera. J Bone Joint Surg. 1988;70A:1224-1230.
- 26 Morrison DS, Bigliani LU. The clinical significance of variations in acromial morphology. Orthop Trans. 1987;11:234.
- 27 Sakar K, Taine W, Uhthoff HK. The ultrastructure of the coracoacromial ligament in patients with chronic impingement syndrome. Clin Orthop. 1990;254:49-54.

- 28 Petersson CJ, Gentz CF. Ruptures of the supraspinatus tendon - the significance of distally pointing acromioclavicular osteophytes. Clin Orthop. 1983;174:143-148.
- 29 Gerber C, Terrier F, Ganz R. The role of the coracoid process in the chronic impingement syndrome. J Bone Joint Surg. 1985;67B:703-708.
- 30 Warner JP, Micheli LJ, Arslanian LE, Kennedy J, Kennedy R. Patterns of flexibility, laxity and strength in normal shoulders and shoulders with instability and impingement. Am J Sports Med. 1990;18:366-374.
- 31 Codman EA. Cited by: Neviasser RJ, Neviasser TJ. Observations on impingement. Clin Orthop. 1990;254:60-63.
- 32 Moseley HF, Goldie I. The arterial pattern of the rotator cuff of the shoulder. J Bone Joint Surg. 1963;45B:780-789.
- 33 Rothman RH, Parke WW. The vascular anatomy of the rotator cuff. Clin Orthop. 1965;41:176-186.
- 34 Rathbun JB, Macnab I. The microvascular pattern of the rotator cuff. J Bone Joint Surg. 1970;52B:540-553.
- 35 Lohr JF, Uhthoff HK. The microvascular pattern of the supraspinatus tendon. Clin Orthop. 1990;254:35-38.
- 36 Chansky HA, Iannotti JP. The vascularity of the rotator cuff. Clin Sports Med. 1991;10:807-821.
- 37 Swiontkowski MF, Iannotti JP, Boulas HJ, Esterhai JL. Intraoperative assessment of rotator cuff vascularity using laser doppler flowmetry. In: Post M, Morrey BF, Hawkins RJ, eds. Surgery of the Shoulder. St. Louis, Mo: Mosby Yearbook; 1990:208-212.
- 38 Neviasser RJ, Neviasser TJ, Neviasser JS. Concurrant rupture of the rotator cuff and anterior shoulder dislocation of the shoulder in the older patient. J Bone Joint Surg. 1988;70A:1308-1311.
- 39 Richardson AB. Overuse syndromes in baseball, tennis, gymnastics, and swimming. Clin Sports Med. 1983;2:379-389.
- 40 Noah J, Gidumal R. Rotator cuff injuries in the throwing athlete. Orthop Rev. 1988;17:1091-1096.
- 41 Clancy WG. Symposium - shoulder problems in overhead overuse sports. Am J Sports Med. 1979;7:138-144.

- 42 Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. *Am J Sports Med.* 1980;8:151-158.
- 43 Jobe FW, Bradley JP. Rotator cuff injuries in baseball-prevention and rehabilitation. *Sports Med.* 1988;6:378-387.
- 44 Fu FH, Harner CD, Klein AH. Shoulder impingement syndrome - a critical review. *Clin Orthop.* 1991;269:162-173.
- 45 Abrams JS. Special shoulder problems in the throwing athlete: pathology, diagnosis, and nonoperative management. *Clin Sports Med.* 1991;10:839-861.
- 46 Uhthoff HK, Hammond DI, Sarkar K, Hooper GJ, Papoff WJ. The role of the coracoacromial ligament in the impingement syndrome. *Int Orthop.* 1988;12:97-104.
- 47 Nirschl RP. Cited by: Fu FH, Harner CD, Klein AH. Shoulder impingement syndrome - a critical review. *Clin Orthop.* 1991;269:162-173.
- 48 Bowling RW, Rockar PA, Erhard R. Examination of the shoulder complex. *Phys Ther.* 1986;66:1866-1878.
- 49 Yocum LA. Assessing the shoulder - history, physical examination, differential diagnosis and special tests used. *Clin Sports Med.* 1983;2:281-288.
- 50 Jobe FW, Bradley JP. The diagnosis and nonoperative treatment of shoulder injuries in athletes. *Clin Sports Med.* 1989;8:419-437.
- 51 Cyriax J, Cyriax P. *Illustrated Manual of Orthopaedic Medicine.* London, England: Butterworth and Co Ltd; 1983: chap 3.
- 52 Nicholson GG. Rehabilitation of common shoulder injuries. *Clin Sports Med.* 1989;8:633-655.
- 53 Cyriax J. *Textbook of Orthopaedic Medicine: Diagnosis of Soft Tissue Lesions.* 7th ed. New York, NY: Macmillan Publishing Co; 1978:chap 9.
- 54 Neviasser RJ. Painful conditions affecting the shoulder. *Clin Orthop.* 1983;173:63-69.
- 55 Pappas AM, Zawacki RM, McCarthy CF. Rehabilitation of the pitching shoulder. *Am J Sports Med.* 1985;13:223-235.

- 56 Inman VT, Saunders M, Abbott LC. Observations on the function of the shoulder joint. *J Bone Joint Surg.* 1944; 26:1-30.
- 57 Watson K. Impingement and rotator cuff lesions. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, MO: CV Mosby; 1990;chap 8.
- 58 Ellenbecker TS, Derscheid GL. Rehabilitation of overuse injuries of the shoulder. *Clin Sports Med.* 1989;8:583-603.
- 59 Bradley JP, Tibone JE. Electromyographic analysis of muscle action about the shoulder. *Clin Sports Med.* 1991; 10:789-805.
- 60 Ivey FM, Calhoun JH, Rusche K, Beirschenk J. Normal values for isokinetic testing of shoulder strength. *Med Sci Sports Ex.* 1984;16:127. Abstract.
- 61 Schieb JS. Diagnosis and rehabilitation of the shoulder impingement syndrome in the overhand and throwing athlete. *Rheum Dis Clin North Am.* 1990;16:971-988.
- 62 Hoppenfeld S. *Physical Examination of the Spine and Extremities.* New York, NY: Appleton-Century-Crofts; 1976: chap 1.
- 63 Neer CS, Welsh RP. The shoulder in sports. *Orthop Clin North Am.* 1977;8:583-591.
- 64 Jobe FW, Kvitne RS. Shoulder pain in the overhand or throwing athlete - the relationship of anterior instability and rotator cuff impingement. *Orthop Rev.* 1989;18:963-975.
- 65 Maitland GD. *Peripheral Manipulation.* 2nd ed. Boston, MA: Butterworths Publishers; 1983:66-67.
- 66 Ogilvie-Harris DJ, Wiley AM, Sattarian J. Failed acromioplasty for impingement syndrome. *J Bone Joint Surg.* 1990;72B:1070-1072.
- 67 Mendoza F, Nicholas JA, Sands A. Principles of shoulder rehabilitation in the athlete. In: Nicholas JA, Hershman EB, eds. *The Upper Extremity in Sports Medicine.* St. Louis, MO: CV Mosby; 1990:chap 11.
- 68 Nitz A. Physical therapy management of the shoulder. *Phys Ther.* 1986;66:1912-1919.

- 69 Moynes DR. Prevention of injury to the shoulder through exercises and therapy. Clin Sports Med. 1983;2:413-422.
- 70 Rocks JA. Intrinsic shoulder pain syndrome - rationale for heating and cooling in treatment. Phys Ther. 1979;59:153-159.
- 71 Downing DS, Weinstein A. Ultrasound therapy of subacromial bursitis - a double blind study. Phys Ther. 1986;66:194-199.
- 72 Wallace L. Personal Communication. (audiotape conversation) August 1992.
- 73 Erb K. Personal Communication. (letter) September 1992.
- 74 Penny JN, Smith C. The prevention and treatment of swimmer's shoulder. Can J Appl Spt Sci. 1980;5:195-202.
- 75 Silliman JF, Hawkins RJ. Current concepts and recent advances in the athlete's shoulder. Clin Sports Med. 1991;10:693-705.
- 76 Hageman CE, Lehman RC. Stretching, strengthening, and conditioning for the competitive tennis player. Clin Sports Med. 1988;7:211-222.
- 77 Townsend H, Jobe FW, Pink M, Perry J. Electromyographic analysis of the glenohumeral muscles during a baseball rehabilitation program. Am J Sports Med. 1991;19:264-272.
- 78 Nuber GW, Jobe FW, Perry J, Moynes DR, Antonelli D. Fine wire electromyography analysis of muscles of the shoulder during swimming. Am J Sports Med. 1986;14:7-11.
- 79 Moseley JB, Jobe FW, Pink M, Perry J, Tibone J. EMG analysis of the scapular muscles during a shoulder rehabilitation program. Am J Sports Med. 1992;20:128-134.
- 80 Anderson L, Rush R, Shearer L, Hughes CJ. The effects of a Theraband exercise program on shoulder internal rotation strength. Phys Ther. 1992;72(suppl):S40. Abstract.
- 81 Warner JP. Personal communication. (letter) July 1992.
- 82 Belle RM, Hawkins RJ. Dynamic electromyographic analysis of the shoulder muscles during rotational and scapular strengthening exercises. In: Post M, Morrey BF, Hawkins RJ, eds. Surgery of the Shoulder. St. Louis, MO: Mosby Yearbook; 1990:32-35.

- 83 Cook EE, Gray VL, Savinar-Nogue E, Medicros J. Shoulder antagonistic strength ratios: a comparison between college-level baseball pitchers and nonpitchers. J Orthop Sports Phys Ther. 1987;8:451-461.
- 84 Ellenbecker TS, DeHart RL, Boekman R. Isokinetic shoulder strength of the rotator cuff in professional baseball pitchers. Phys Ther. 1992;72(suppl):S81. Abstract.
- 85 Ellenbecker TS, Davies GJ, Rowinski MJ. Concentric versus eccentric isokinetic strengthening of the rotator cuff - objective data versus functional test. Am J Sports Med. 1988;16:64-69.
- 86 Janda DH, Loubert P. A preventative program focusing on the glenohumeral joint. Clin Sports Med. 1991;10:955-971.
- 87 Hawkins RJ, Brock RM, Abrams JS, Hobeika P. Acromioplasty for impingement with an intact rotator cuff. J Bone Joint Surg. 1988;70B:795-797.
- 88 Wasilewski SA, Frankl U. Rotator cuff pathology. Clin Orthop. 1991;267:65-69.